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THE APPROACH TO THE STUDY OF HYSTERIA.*

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The vicissitudes undergone by hysteria in the succession of the centuries have varied prodigiously, and he would be a bold clinician who to-day would announce their definitive end. Phases of medical doctrine run their course and have their day, but hysteria goes on. Doubtless in the present era of psychological grace we who have been conscious of the immense strides taken by psychopathological research are prone to believe, if not, perhaps, to feel entirely sure, that hysteria has at length yielded its secret—a discovery often heralded before, but never, we fain would pride ourselves, with so valid a pathogenesis on which to base the claim. If, for the nonce, however, we approach the question from the standpoint of pure empiricism, confidence may conceivably be somewhat shaken.

To this empirical approach objection cannot be raised. No reason exists why study of the objective clinical phenomena of the condition should not be prosecuted, deductions therefrom as to their nature drawn, and generalizations of a nosological character formulated, or, at least, attempted. Nothing, however, has been more typical of this period of psychological study, so far as hysteria is concerned, than the comparative absence of research of an objective and empirical kind. Can it be that acceptance of the former method dispenses with all necessity to conduct the latter ? If this be the case, then psychological theory stands to lose in impressiveness. It is not so very long ago since a distinguished protagonist of new conceptions of hysteria, in a psychiatry club of which I was a member, listened with mild impatience to my exposition of certain neurophysiological peculiarities in hysterical symptoms, and closed discussion by declaring that since the neurosis had its demonstrable origin in a vita sexualis which did not run smooth its semeiology was of merely secondary interest and minor importance.

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Possibly the outstanding feature of hysteria as revealed to us by the records of former generations and the knowledge of our own is the changes which its clinical syndromes have suffered. This simple fact of observation must appear highly significant, as fruitful for theory as it is true in substance. The mediæval ecstatic, simulating in hands and feet the nail-prints of her Redeemer, is long since démodée, as is her eighteenth-century sister, melodramatically counterfeiting the emotional transports of a popular actress. I quote from Kay's *Portraits*, published in 1837 :—

> At the first visit of the great Mrs. Siddons to Edinburgh, many were the fainting and hysterical fits among the fairer portion of the audience. Indeed, they were so common that to be supposed to have escaped might almost have argued a want of proper feeling. One night when the house had been thrown into confusion by repeated scenes of this kind, and when Mr. Wood (a popular surgeon) was most reluctantly getting from the pit (the favourite resort of all the theatrical critics of that day) to attend some fashionable female, a friend said to him in passing, "This is glorious acting, Sandy," alluding to Mrs. Siddons ; to which Mr. Wood answered, "Yes, and a damned deal o't, too," looking round at the fainting and screaming ladies in the boxes.

What impresses in this echo from an almost unimaginable past is the insight displayed by the common-sense surgeon of that day. But let us come a little nearer our own time. The quotation is from a lecture on hysteria delivered at St. Bartholomew's Hospital in 1866, by Mr. F. C. Skey, F.R.S., a then celebrated member of its consulting staff :—

> Thirty or forty years since . . . and for how many years anterior I know not, all the seaside towns were crowded with young ladies between 17 and 25 years of age and beyond it, who were confined to the horizontal posture, and wheeled about on the shore in bath chairs, on the supposition that they were the subjects of spinal disease. They were placed under much medical and dietetic discipline . . . and the large majority carried a pair of handsome issues in the back ! Brighton, Worthing, Hastings, and other places on the South Coast were largely tenanted by these unfortunate females, to which a modest sprinkling of young gentlemen was attached. What has become of all these cases ? They appear to have vanished just in proportion as the eyes of the surgeon have been opened to the absurdity of inferring that pain alone, which locates itself with remarkable precision in hysteria on a given vertebra, can indicate the presence of organic disease of the body of the bone without collateral evidence in its favour.

No longer do the ' circus horses ' of the Salpêtrière perform before visitors as in the palmy days of Charcot ; no more does their contorted musculature respond to the application of divers metallic rods, as Gilles de la Tourette was wont to demonstrate ; seldom indeed is the clinician witness to the elaborate and protracted hysterical fits whose theatrical features were drawn with artistic skill by Paul Richer. The intimate correspondence of the historical phases of hysteria with contemporary medical predilections is responsible for the alternate tragedies and comedies of its treatment, and Morestin's remark embodies a shrewd estimate of the situation : " Les hystériques suivent à leur manière le mouvement scientifique, et s'adaptent au progrès de la médecine." Since the day (and long before it) of the insensitive and bloodless marks that stamped the witch as Satan's own the poor hysteric has done her best, has

never failed to respond to the calls made on her. But to-day we seek the clue to the ailment in the unseen psyche, and she is somewhat at a loss accordingly ; her elaborated somatic manifestations are rather at a discount. A cold scientific environment besets her, instead of a world of emotional extravagance and limitless credulity. So a defence-hysteria is now à la mode, no less egocentric if more utilitarian ; with an eye to the main chance, the hysteric turns to the possibility of compensation for trauma, or seeks to escape from unpleasant reality by a flight into neurosis—at least, so we are assured. The times have changed, and we, both physicians and hysterics, have changed with them.

Yet, when all this is said, it remains true in a deeper sense that if hysterical fashions vary, their kaleidoscopic patterns are composed of unaltering, instinctive and impulsive reactions ; thus can its clinical characters be recognized on the Greek vase depicting the ecstasies of the frenzied mænad, in the records of some New Testament miracles, in mediæval paintings of demoniac exorcism, in modern accounts of camp-meetings, spiritualistic séances and religious revivals, of disease-mimicry in school-girls and shell-shock amid twentieth-century warfare. Beneath surface diversities the essence of the phenomena involved has not been modified. And further, where old beliefs linger in the public mind, where superstition is rife and mysticism rampant, there still is to be found the favourable and appropriate milieu for hysterical exhibitions of antiquated form and guise even in this epoch of scientific enlightenment. The literature of demoniacal possession, recently collated and analysed by Oesterreich, contains not a few references of an authentic kind belonging to the last two decades only. Nor, in the nature of things, is it likely that such hysterical traits and expressions will really disappear ; human culture may progress, but a Messina earthquake or a world war will lay bare hidden reactions among some of the species, at all events. Leprosy, rabies and malaria are ancient diseases, but epilepsy and hysteria are older still ; are they as old as human nature itself ? Their antiquity and ubiquity might almost suggest this ; and since epilepsy is scarcely anything else than a physiological reaction, which anyone soever will exhibit provided he is injected with a convulsant, it becomes curiously interesting to discuss whether hysteria, too, is but a name for a reaction, capable of being expressed in dynamic, neurophysiological terms.

It is nearly twenty years since I wrote a paper in *Brain* on this subject, in which certain ideas respecting the objective study of hysterical phenomena were outlined.

A brief quotation will indicate what is meant :—

> I believe we must depend on the objective signs of hysterical disease if we are to progress towards its unification. Janet himself holds that the intrinsic examination of cases of hysteria is the most accurate and scientific method. Now the chief point, as it seems to me, on which more light is wanted, is the mechanism of production of the symptoms. Granted that a particular mental state is accountable for the disturbances of function, how are these actually brought into being ? Granted

> that amnesia or absentmindedness [in accordance with Janet's views then being examined] causes hysterical motor defects, why have we tremor in one instance and paralysis in another? And what are the objective features of these? Can we thereby learn anything as to the seat of the functional defect? I am convinced from the study of a number of cases that organic disease will teach us far more about hysteria than vice versa.

Of course, the physiological approach is only one of several. And in directing attention thereto for the moment I do not ignore that which seeks to utilize as much—as exclusively—as possible the assumption of 'unconscious mental processes' and of their influence on somatic pathways; nor that other which surveys the hysterical field from the standpoint of causation by more or less conscious processes of simulation. But it is permissible to examine the data without subscribing to any particular theory, and to ascertain whither such study leads. That physiological investigation is legitimate needs no apology whatever; both in former and latter times clinicians have busied themselves therewith, but few if any psychologists—with one noteworthy exception to be referred to later. Kretschmer's recent analysis of hysteria is concerned to a large extent with physiological considerations, though not at all on the lines now to be sketched.

The method chosen no doubt depends on the scientific taste and orientation of the individual physician, and I confess to having been influenced by a teacher who besought me in neurological work never to 'invert the pyramid.' For a number of years clinico-physiological observation of hysterical phenomena has occupied my time at intervals, and a few of the collected data may now be passed in brief review and their bearing on major hysterical problems examined.

MOTOR SYSTEM.

Hysterical paralysis is a clinical banality, but study of its intrinsic characters will be found illuminating. It assumes the form of paralysis in rigidity, or of paralysis in flaccidity. Otherwise expressed, the muscular weakness may or may not be accompanied by plain alterations in muscle tonus. When weakness or paralysis is associated with increased tone the condition clinically is usually, perhaps invariably, one of contracture. In flaccid cases the part affected seems emptied of tone, though not always perhaps to the degree met with in atrophic lower motor neurone affections, such as poliomyelitis. All intermediate stages between the 'toneless-flaccidity' and 'spasmodic-rigidity' extremes may be encountered.

So far as I can discover, my former chief, the late Dr. C. E. Beevor, was one of the first to direct attention to the occurrence in hysterical palsies of the phenomenon known as 'defective inhibition of the antagonists.' He had under observation the case of a girl aged 18 with incomplete right hysterical hemiplegia, and noted that whenever she was asked to perform a certain movement the first action detected was that of the antagonist muscles. For instance, on being told to extend the elbow, the first muscle observed to contract was the supinator longus, one of the antagonists to the movement of extension. This

was followed immediately by that of the triceps, and there succeeded a confusion of to-and-fro movements due to alternate or simultaneous contraction of extensors and flexors of the elbow. When she was requested to flex that joint the triceps sprang into contraction first. I was Dr. Beevor's house physician at the time, was duly impressed with the basic significance of this phenomenon for hysteria, and have made it a matter of routine investigation ever since. It is unquestionably the most common motor sign of the affection and can usually be demonstrated with ease. It underlies the vast majority of hysterical disturbances of active motility, being equally remarked in contractures ; when the patient seeks to undo a contractured attitude the antagonists will be found to contract still further.

Beevor declared in his Croonian Lectures (1904) that he had never met with this condition of the antagonists acting before the prime movers except in hysterical paralysis, and thought it a diagnostic feature of that state. Oppenheim also admits its frequency under such circumstances.

The import of this physiological disorder resides in the fact of its obviously constituting a disturbance of the Sherringtonian law of reciprocal innervation, according to which as a given muscle contracts in a normal movement its antagonist is simultaneously de-innervated. In the experimental animal reciprocal innervation holds for all spinal and, more generally, decerebrate reflexes. But Beevor's view of its pathognomonic value for hysteria, though not discounted, must be considered in the light of another fact, viz., that, as was shown in my Croonian Lectures (1925), the phenomenon occurs also in athetosis. In that 'organic' affection the orderly innervation and de-innervation of prime movers and antagonists are interfered with. Further, and still more significantly, an endeavour was made in these lectures to show that athetosis results from disordered function of certain *cortical* reflex arcs, and that the underlying lesion *may* be of cortical site. Another condition attended by precisely the same physiological defect, as I have observed, depends on lesions of the superior frontal cortex sometimes associated with the symptom known as 'tonic innervation' and with apraxia. Thus it appears to be of deep interest for the study of hysterical physiology that a special symptom distinguishing it with such constancy is also met with in one or two 'organic' states whose site is cortical ; and the inference seems fully warranted that hysterical palsies bear the physiological imprint of cortical defect of function.

A conclusion of this kind may appear à priori obvious from the standpoint of theory, but to have reached it on objective considerations alone is surely satisfactory, the more so as it furnishes an instance of explanation of hysterical physiology from the 'organic' side.

Another no less interesting motor sign consists in the shunting of the desired innervation to other groups of muscles, not confined to the actual antagonists. This is well seen, for example, in hysterical paresis of the arm ; on the request to grasp the examiner's fingers tightly the patient gives the

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impression of putting forth a great effort, but the grasp remains feeble, while it is easy to be assured of the fact that shoulder muscles and others equally remote from the long flexors are in strong contraction. This 'shunting' sign, as it may be termed, is highly characteristic of hysteria, but has its analogies in certain states of muscular weakness following structural lesions —yet under these latter circumstances it is seldom or never so pronounced as in the other.

The fact that both of these physiological traits are such as can be imitated by the normal subject has led some observers (v. Hösslin, Bassett-Jones and Llewellyn) to regard the presence of either as proof of simulation, or at least of an attempt to deceive. But in my opinion this view betrays a merely superficial insight into their meaning. The criterion of imitational capability is worth exactly nothing. With practice the normal subject can so empty his musculature of tonus that a limb can become as flaccid as in poliomyelitic paralysis—indeed, the detonization may proceed so far as to result in actual loss of the knee-jerk, as Jacobson has conclusively shown. Here is an 'organic' condition—loss of deep reflexes—which can be effected temporarily by the aid of 'volition' (I regret introducing that highly ambiguous word at this stage, and shall have something more to say of it later). Where, then, is the value of imitation as proof that hysteria and simulation are akin or identical? One might as legitimately claim that atony with loss of a tendon reflex is hysterical and evidence of malingering, since it is not outside the faculty of normal man to reproduce it. The variety artiste on the stage can so deplete his muscles of tone as to give an excellent rendering of a dummy figure, a jointless doll—as atonic and as seemingly 'paralytic' as any state of 'organic' disease could ever be. With practice, again, a very fair reproduction of some of the salient features of Jacksonian epilepsy can be made.

The real point, I submit, is that in respect of motor phenomena imitability characterizes more especially those whose physiological seat is cortical, and that symptoms of escape or release at lower levels can be reproduced with perhaps less facility. However this may be—and it is undesirable at present to lay any stress on the generalization—both the 'shunting' and the 'antagonist contraction' signs indicate, as is here contended, impairment of cortical function, coupled with defective inhibition. The hysteric cannot inhibit antagonistic innervation, nor can he prevent displacement of innervating currents in an erroneous direction.

A word, next, on those forms of motor disability which consist of 'positive' movements. Hysterical tremor, for instance, considered physiologically, presents no separate or contrasting features when set alongside those of so-called 'organic' type. I have frequently found intention-tremor clinically indistinguishable from that of disseminated sclerosis. The reputed differentia of variability, of influence by psychical and emotional stimuli, of dependence on the attention paid to it, and others, are demonstrably invalid. Numerous 'organic' tremors can be affected by a whole series of factors, exhibit marked

fluctuation and fluidity, are highly irregular, shift their incidence, and are aggravated when the subject is under observation. A hundred years ago James Parkinson himself noted the disappearance of the tremor of paralysis agitans in consequence of mere change of posture. As a matter of observation, not a few hysterical movements of this kind sometimes seem as fixed and as invariable as those deemed the result of structural lesions. Now it is within common knowledge that hysterical subjects exhibiting such movements do not complain of fatigue, or at least appear to be less conscious of it than does a normal subject who executes them intentionally; moreover, the Parkinsonian and the torticollic, by way of instance, frequently (though not constantly) admit muscular fatigue and aching. Kretschmer, I consider, is mistaken in asserting that "a rapid, rhythmical and somewhat constant shaking tremor, in general, will absolutely not allow itself to be imitated voluntarily under normal experimental requirements, either by healthy or by nervous persons." On the contrary, we can easily convince ourselves of the possibility of imitation by actual practice. But he rightly points out that under the latter circumstances it leads to such a degree of fatigue that its continuance becomes impossible. The difference in this respect seems to depend on the fact that in hysteria, as in 'organic' states, tremor is an escape-phenomenon of an infracortical level, and that the experimenter is presumably utilizing neomotor mechanisms to reproduce the activities inherent in palæomotor mechanisms of a lower physiological grade; fatigue sets in more readily in the former case because such artefactive tremor forms no part of their habitual function. At any rate, it seems feasible from a physiological point of view to attribute the 'positive' movements of hysteria to failure of transcortical inhibition when their type is high, and of corticifugal inhibition when their type is low.

So far as the multiplex syndromes categorized as the hysterical fit are concerned, their range from fugues at the one extreme to immobility and lifelessness at the other covers so wide a symptomatic field as perhaps to render attempts at synthesis precarious. Reduced, however, to their lowest terms, one and all are indicative of release of function from conscious control. Differences between hysterical and epileptic fits are matters of degree and not of kind. In each, the depth to which unconsciousness proceeds is variable, and if more profound in the average major epileptic seizure may be serviceable in clinical diagnosis but not in nosological differentiation. Both show convulsive and quasi-purposive movements on occasion, the latter predominating in hysteria; and while cyanosis, tongue-biting, and micturition frequently distinguish the one, and automatisms the other, the distinctions resolve themselves into the simple question of which mechanisms are prone to be involved, and which to escape, in either. Otherwise expressed, clinical differences stand for modifications in site and extent, but not for intrinsically or essentially different processes. A general resemblance between the attitude in extensor rigidity of many hysterical attacks and that of the decerebrate animal cannot be denied, for it is a matter of direct observation; moreover, it is to be expected, since

if for any reason, through any agency, neuronic control of infracortical mechanisms is in abeyance, these are bound to exhibit on release their specific functional qualities. The physiological characters of some hysterical fits may be taken as evidence of transient (perhaps incomplete) decortication or decerebration, nor can I think any other interpretative view permissible. For the student of hysterical physiology the problem is to examine and determine to what extent, down to what levels, neuronic dissociation can be effected by the processes of hysteria.

SENSORY SYSTEM.

The empiricist taking the sensory derangements of hysteria at their face value notes the neuralgias and topalgias on the one hand, and the anæsthesias on the other. He is struck by their variety and variability, their capacity for rapid change, and—so opinion has it—their failure to correspond to anatomical schemes: they are alleged to conform with psychical but not physiological dispositions. As illustration of this latter statement we see cases in which the breast but not the nipple is anæsthetic, the front of the body but not the back, and we are familiar with the 'glove' and 'stocking' anæsthesia of the textbooks.

But ere assuming therefrom that hysteria is a 'disease of the mind,' a disorder on the plane of mental images, we do well to recall that the physiological arrangement of the mechanisms underlying conduction of sensory impulses varies at successive neural stages. Peripheral loss of sensibility can be distinguished from spinal loss, spinal from thalamic, and thalamic from cortical. In respect of the last of these the clinician can prove from actual experience that a re-arrangement or re-representation of lower sensory schemata takes place, and that with some forms of cortical lesion the distal parts of the opposite limbs are much more affected than the proximal. This fact of itself at once reduces the differential value of 'glove' and 'stocking' anæsthesia, apart from the other fact that it may be found in syringomyelia, as was demonstrated years ago by Brissaud. Further, as regards the arm more especially, evidence can be adduced which clearly suggests the existence in the cortex of a pre-axial and post-axial segmentation, and errors of reference in either direction can be shown to occur with certain cortical lesions. A front-and-back schematization is not only inherently possible, but is rendered probable by studies of Redlich, Vierheller, and others, in both spinal and prespinal cases 'organic' in origin. We recognize the existence of 'sidedness' in sensory disorder of the highest level, and that on an 'organic' basis: allocheiria or, preferably, dyscheiria, considered distinctive of hysterical states, I have seen characteristically in disseminated sclerosis. Some evidence has accumulated favouring the conception of what may be called a longitudinal segmentation of the trunk in the cortex.

We need not, however, enter at large into details of this kind. The point that emerges is simply this, that in the light of such data it is rash to presume a psychogenic basis for sensory disorders because of peculiarities in their seg-

mental type when there is presumptive evidence of physiological arrangements in the cortex to explain their occurrence.

Some reference is here desirable to the not infrequent concomitance of hysterical disturbances of sensation with absence of bleeding when the cutaneous zone affected is pricked with a pin. So ancient and notorious an observation as this becomes unusually instructive when set beside a further clinical fact, viz., that precisely the same combination has been seen in connec ion with 'organic' sensory change caused by a lesion in the formatio reticularis of the medulla. I have reported elsewhere the analysis of a case of occlusion of the left posterior inferior cerebellar artery in which the patient discovered for himself that he did not bleed in the analgesic area when the skin was punctured by a needle. I quote from the original record :

> Mr. M. had often noticed that pricking of the left face, in addition to its being felt as pressure merely, never resulted in any bleeding. He had frequently experimented with himself in this connection, and says the same thing is true of the right side of the body. About three weeks ago, however, coincident with the diminution of the subjective sensations on the left face, he found that pricking was followed by bleeding for the first time since the onset. I re-examined him from this point of view, and can vouch for the fact that blood is much more readily obtained by pricking on the left hand than on the right, whereas there is no difference now between the two sides of the face.

That some vasomotor change had occurred over the left face and right body and limbs seems certain—presumably a degree of vasoconstriction. The association of the same vasomotor state with hysterical anæsthesia is deeply interesting. I remember a case of complete hysterical paraplegia in a young woman of 22 where the degree of analgesia was so profound that a needle was passed through the calf without its being perceived, and without causing any bleeding. After a single application of the wire-brush the analgesia disappeared and bleeding occurred at once whenever the skin was pricked.

It seems only one conclusion is permitted by comparison of these two cases ; an alleged stigma of hysteria turns out to occur also from 'organic' lesions of the medulla and loses any specificity it is thought to possess ; moreover, the inference can justifiably be drawn, I submit, that bulbar centres and mechanisms are involved in certain cases of hysteria, and that these in part belong to the neurosympathetic system.

REFLEX SYSTEM.

Anyone with clinical experience will allow that reflectivity frequently exhibits departures from what is normal. In hysterical hemiplegia or paraplegia the observation is now old and well-established that the plantar response is often lost : with repeated stimulation no reaction is obtainable, and this, too, when the foot is neither specially cold nor anæsthetic. I have been familiar with this for many years, as also with the fact that the abdominal reflex may be reduced (or even lost) in hemiparetic cases, usually in association with sensory diminution on the same side—an observation substantiated by

Monrad-Krohn. Of the occurrence of hysterical ankle clonus no doubt can be entertained. Interference with sphincteric action, too, needs no comment; incontinence of urine was present for six weeks in the case of a hysterical paraplegic under my care, and retention is no rarity.

Dr. C. S. Myers is the psychologist referred to above who, almost alone of his class, has paid any attention to these phenomena, or evinced appreciation of their importance. At the end of the War he said that "in this country at least we have been paying so much attention to the mental aspect of the war neuroses that a detailed examination of the accompanying bodily symptoms has been generally neglected." His own observations showed him the frequency with which the plantar reflex fails, and he also remarked in many cases of asymmetry as regards the two that both knee-jerk and abdominal reflex appeared weaker on the same side as the feeble flexor or abolished plantar response, and that diminished appreciation of cutaneous stimuli was usually in evidence also.

I long ago satisfied myself that distinctions between 'organic' and hysterical ankle clonus formerly relied on could not be trusted: Babinski's criterion that the former is present each time the patient is examined, whereas the latter varies and may not be elicited on occasion, has, from my personal experience, turned ont to be valueless. Nor does irregularity of rhythm carry any differential weight. Now if diminution of the abdominal reflex is depended on as an early sign in frontal tumours, disseminated sclerosis, and pyramidal conditions generally, I cannot see why the same phenomenon in cases of hysteria should not bear the same meaning or import. It is unnecessary now to advert to the rather disputed question of the development of an extensor plantar reflex in hysteria, but loss of the plantar reaction must signify some physiological change. A similar reduction characterizes many cases of 'organic' hemiplegia at the acute stage, and according to Ganault the reflex is completely abolished in 10 per cent. No reason whatever can be advanced for accepting these physiological specialties in 'organic' affections and denying them equal significance in cases of hysteria.

For simplicity's sake a few comparatively common, every-day symptoms of hysteria have been selected for examination from a neurophysiological angle. This sketchy and compulsorily limited review has perhaps sufficed to demonstrate how cortical, infracortical, mesencephalic, bulbar, spinal and neurosympathetic mechanisms may severally be disordered in function as a result of processes which we call hysterical. No doubt in one sense this is a mere platitude: for after all what others are there for hysteria to usurp than those of the person concerned? But it is something, surely, to be able to show that differentiating criteria as between organic and hysterical conditions have lost much of their specificity, that symptoms thought exclusively hysterical

reappear under circumstances never assigned to the interference of the psyche, and that many of the somatic manifestations of that 'neurosis' have their counterpart in known physiological derangements of one grade or another. In one at least of its aspects the affection becomes less mysterious when its activities can be traced through different physiological levels.

The progress of medical knowledge has been so gravely hampered by persistent use of the two terms 'organic' and 'functional' that it is high time they should be discarded for ever. Scarcely less objectionable is the alternative antithesis of 'structural' and 'nutritional.' (I have studiously avoided in this discussion the word 'functional' even as reputedly synonymous with 'hysterical.') In their place, if terminology is required, we may with advantage employ the pair of opposites 'static' and 'dynamic.' It must be clear on due consideration that innumerable nervous symptoms stand for dynamic alterations in function which cannot at present be correlated with any recognizable lesions. In the case of a major epileptic fit manifesting itself all over the body, so to speak, no one can imagine for a moment that there are corresponding 'lesions' all over the nervous system. The display is a physiological reaction to some nociceptive excitant whose site of initial action is probably extremely limited, but a dynamic, and transient, extension follows over a wide region of normal nervous tissue. In a number of disease-conditions associated with static lesions the symptoms themselves exhibit fluidity of the most obvious kind, as has already been alluded to in connexion with the tremors of paralysis agitans. How then can we link them to the cellular changes we detect under the microscope?

Applying this general semeiological conception to hysteria, we note at once the prominence of the dynamic and the comparative absence of any static elements therein. We are impressed by the strictly physiological inhibitions and excitations of that state and by their ranging over higher and lower mechanisms alike: but at the same time their essentially unfixed and fluid character stands out saliently. How can we explain the rapid cure of hysterical disabilities—or shall I say their clinical disappearance—how understand that nervous mechanisms are as it were ready to resume duty at a moment's notice, after perhaps some years of disuse, unless on the hypothesis that function is abrogated dynamically but not statically? There is a block somewhere, a pure inhibition, in the case of negative symptoms: or, on the other hand, inhibition is removed and release-phenomena come into being as positive symptoms. Neither can be correlated with lesions of a static kind, but that physiological derangement of a dynamic character is invariably present seems almost self-evident, and from the illustrations already furnished may be considered objectively proved. The difference, then, from so-called 'organic' conditions does not reside in the co-existence of static change, for this need not accompany all the manifestations of that class, but rather in the manner of onset of the symptoms and the manner of their removal.

ETIOLOGY.

We can trace through the symptomatological diversities of hysteria the action of ordinary physiological processes, precisely the same as are encountered in the dynamic symptoms of affections we categorize as having a basis in recognizable disease of tissues: but as soon as we approach the question of their origin and pathogenesis difficulties accumulate. In these days a majority of psychiatrists and neurologists presumably adopt the view of their ideogenic origin, and regard this as almost axiomatic. Perhaps the most commonly accepted theory is that they are conditioned by one or other kind of sexual conflict, of which, to take concrete examples, some may be enumerated as follows: jilting in love, jealousy, lovers' quarrels, a sister's marriage, fear of pregnancy, marital unhappiness and so forth. To enlarge the catalogue would be both easy and superfluous. Frankly, this strictly sexual etiology appears incapable of universal application to questions of hysterical pathogenesis. Why and how such psychical incidents should produce loss of the plantar reflex, or local vasoconstriction and cutaneous anæsthesia, eludes comprehension. On the view that hysterical symptoms are nothing else than reactions to stimuli, it still is difficult to understand why psychical excitations of this completely restricted sexual kind should be capable by themselves of evoking responses ranging from fugues to retention of urine. With some knowledge of the extensive psychological literature on the subject I have not met with any serious effort amid its profuseness to correlate etiology and symptomatology. Between the one and the other stretches a pathogenic hiatus of a glaring kind, which cannot be said to facilitate acceptance of the sexual theory.

If the etiological net is cast a little wider, we might adopt the hypothesis that hysterical reactions are determined by the emotional life in an unrestricted sense. Emotional experiences and impulses, originating in whatever fashion, associated with sexuality or not, do unquestionably help to engender the development of hysterical syndromes: they may be taken as exciting, if not predisposing, causes. It is, perhaps, easier for the trained neurologist to comprehend the physiological effects of emotional disturbance, since he has at least a little knowledge of the mechanisms of the neuraxis underlying the expression of the emotions. Radiation of emotional effect can be translated into anatomo-physiological terms: the somatic and neurosympathetic accompaniments of emotional action range widely and are capable of objectivation. Varieties of shock have their reverberation in known high and low level neuronic systems. Nevertheless individual hysterical reactions, monosymptomatic types, are not easily correlated with the causative action of so *general* an excitant or inhibitant as emotion. Of course I recognize and for that matter have myself observed the occurrence of hysterical symptoms (such as aphonia or amaurosis, for example) in which it has been feasible to link the *form* of the reaction to some pre-existing incident connected with the particular part of the body affected, but in numerous others it is impossible to trace any such

association. A comprehensive study of hysterical semeiological types in relation to etiological variants is urgently needed.

The assertion that we are all potential hysterics, often put forward both before the War and since, is explained by Kretschmer to mean that everyone carries within him old, instinctive, primitive forms of reaction more or less covered up and controlled by the acquisitions of culture. Most of those who have had experience of hysteria will agree that it is apt to appear at certain physiological periods under circumstances of an emotional kind. Yet at times it undoubtedly arises unexpectedly; I have seen hysterical hemiplegia in a King's prizeman at Bisley. To the generalization, however, it is impossible to give formal consent; on the contrary, I submit that the evidence favours the view that the hysteric is born and not made, and that an unstable constitutional background, a 'temperament,' if the word is preferred, is the soil from which hysterical growths spring. And it seems feasible to suggest that this determinant may be in reality of a physiological and not a psychological kind—that it is constituted by an inherent lability of reaction, a lack of neuronic cohesion and firm integration, an abnormal facility in dissociation of neural systems, such as undoubtedly characterize hysterical semeiology from the viewpoint of nervous function. When we are told that the "acquisitions of culture" cover up the "primitive" within us, it appears to me preferable to say that transcortical inhibition dominates the neuraxis: when the hypoboulia of the hysteric, his weak will, is paraded as explanatory of hysterical pathogenesis, a physiological exposition of the data might, in my view, equally well be sought: we might speak rather of failure of one cortical element to inhibit another—a state of affairs the extreme likelihood of the occurrence of which is postulated by Sherrington for various fields of cortical action. That 'volition' seems constantly, or at least repeatedly, to take a share in the production of hysterical symptoms is notorious, and is responsible for the not unnatural view that simulation enters largely therein; but it is conceivable that the physiological basis of the 'will' is inhibition: that a 'voluntary' movement signifies merely one which is transcortically permitted: that 'letting go' rather than 'doing' represents the true meaning of a 'volitional' act. With a conception of this kind, developed along physiological lines, the peculiar features of hysterical motor disorder receive a simple explanation: we do not mix up a psychological impairment—a weak will—with a physiological derangement—defective inhibition of antagonists—but take the former to signify failure of transcortical control and elucidate the latter thereby. An ingenious explanation, on purely physiological grounds, of the phenomena of hysterical palsy was published by Graham Brown and R. M. Stewart in a paper which appeared at the end of the War and seems not to have attracted the attention it certainly merits.

The general tenor of the argument here outlined with some restraint is that physiological processes lie behind nervous manifestations at all levels: that the cortex, considered as a ganglion, cannot differ inherently in function

from other, infracortical, ganglia except as regards complexity: there is no reason to suppose nature makes a jump from one order to another totally different at the cortical stage. Reflex actions are there to be expected, in analogy with those located inferiorly. The present trend of physiological research is to reduce steadily the number of functions once ascribed to 'volition': for example, mechanisms controlling posture and balance are now taken away from it and assigned to reflex arcs independent of the higher levels. So far as the cortex is concerned, I am by no means alone in arguing that much cortical activity partakes absolutely of a reflex character: and I fancy that what for want of a better expression we term the 'will' is being driven from one transcortical ditch to another as it struggles to survive. It seems logically sound to seek to interpret cortical activities in physiological terms alone, and to avoid use of the psychological term 'volition' altogether. Admitting that many of the phenomena of hysteria belong obviously to the highest grade of activity, are so like 'volitional' actions as to be thought indicative of deception or simulation, it is suggested they represent transcortical disorder of function, on the strict analogy of functional release at lower levels. An approach from below upwards, as it were, has much to commend it, for the gap between physiological and psychological systems is scarcely likely to be bridged from the psychical side.

THE PATHOLOGY OF CHRONIC EPIDEMIC ENCEPHALITIS: A HISTOLOGICAL STUDY OF FOUR CASES WITH WIDESPREAD CEREBRAL LESIONS.*

By

ROBERT CARMICHAEL, Edinburgh.

The predominant importance of the Parkinsonian syndrome amongst the later manifestations of epidemic encephalitis has caused a considerable number of observers to direct their efforts towards the finding of an anatomical basis for the development of this symptom-complex. As a result, attention has been focussed upon the substantia nigra, in which severe destructive changes are constantly found in these cases and the rest of the central nervous system is commonly described as showing changes of only slight significance (McAlpine[1,2]). In the present study, on the other hand, attention has been given to the disease-process and its effects upon the nervous tissues as a whole rather than to the results of destructive changes in definite areas.

Two of the four cases which were available for study were of the ordinary Parkinsonian type and the other two were selected because they showed, during life, symptoms of severe cerebral disturbance, compatible with a diagnosis of chronic encephalitis, although not definitely indicative of this condition: and because examination of the brain revealed marked destructive alterations in the substantia nigra and failed to reveal the anatomical changes characteristic of any other recognised disease.

Material and Methods.—The brain and spinal cord were fixed entire in either formalin or formalin-ammonium-bromide solution. The brain-stem was cut transversely into a series of thin slices and each alternate fragment embedded in celloidin, the remainder being used for frozen sections. Pieces of tissue from the pre-frontal, pre- and post-central, occipital and temporal cortex, basal ganglia and thalamus of each hemisphere, from the cerebellum and from the four principal regions of the cord were also cut both in celloidin and after freezing. Celloidin sections, cut at about 15 micra, were stained by Masson's hæmatoxylin-eosin-safran method and by thionin or toluidin blue. Scharlach R. staining for fats, silver impregnations for the demonstration of axis-cylinders, Cajal's gold-sublimate method and Anderson's Victoria-blue technique for the neuroglia were carried out upon frozen sections. Several other methods were also used for special purposes.

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CHANGES IN THE INDIVIDUAL TISSUE ELEMENTS.

A brief account of the alterations in the individual tissue elements will save unnecessary repetition in the description of the individual cases.

Nerve-Cells.—As the localisation of the lesions in the grey matter would lead one to expect, the perikarya of the neurones are commonly severely affected while their axis-cylinders and myelin sheaths usually escape damage. Regressive alterations in the former are frequently observed whereas signs of injury to the latter are rarely noted and, when they do occur, are probably always secondary to the destruction of the cell from which the fibre originates.

In every part of the nervous system the smaller nerve-cells show more conspicuous degenerative changes and are surrounded by larger groups of perineuronal satellites than the larger elements in their neighbourhood. Thus the small stellate and polymorphic cells in the cerebral cortex suffer more severely than the larger pyramidal cells: the small polygonal cells in the lentiform nucleus present more marked alterations than the large pallidal cells: the small cells forming the sensory nuclei of the brain-stem are often severely damaged, while the large multipolar elements of the motor nuclei are little affected.

The extensive destruction of the large multipolar cells of the substantia nigra may suggest that the presence of a considerable quantity of melanin pigment in the cytoplasm makes the cell particularly vulnerable, but outfall of pigmented cells in other regions, notably the substantia ferruginea and dorsal motor nucleus of the vagus, is generally inconspicuous.

The degenerative changes in the cells present no noteworthy peculiarities. Chromatolysis occurs, as in other conditions. The Nissl bodies break up into coarser and then into finer granular fragments, first near the nucleus and later throughout the cell-body and dendrites: the nucleus loses its central position in the cell and becomes slightly swollen: the nuclear membrane stains rather less distinctly while there may be a faint colouration of the intranuclear network: the nucleolus is somewhat reduced in size, loses its sharp contour, becomes vacuolated and stains more feebly.

In more severely damaged cells the cytoplasm and dendrites may take a faint, homogeneous stain and the cell-body and processes appear much distorted, while smaller and larger vacuoles appear in the cytoplasm and, if numerous, give the whole cell a foamy appearance. More commonly the Nissl substance disappears almost entirely from the cytoplasm, the cell loses its clear-cut outline, and a small amount of faintly-staining, granular material which appears to be disintegrating at the periphery is all that is visible around the nucleus.

But even in the presence of the most severe alterations in the cytoplasm of the nerve-cells, changes in the nucleus are seldom more marked than those already described. Complete solution of the nuclear chromatin, fragmentation of the nucleus or nucleolus and extrusion of the nucleus from the body of the cell are all extremely rare phenomena. It seems reasonable to conclude,

therefore, that the majority of the cells which are severely damaged are not necessarily on the way to complete destruction but are, under favourable conditions, capable of recovery.

Granule-Cells.—Cells of this type play a very prominent part in the reactive processes in these cases and are usually found in large numbers clustered about the cell-bodies of the neurones, wandering free in the tissues or lying in rows along the smaller vessels. Their nucleus, which is small, deeply-staining and rounded or slightly ovoid, closely resembles the nucleus of the ordinary

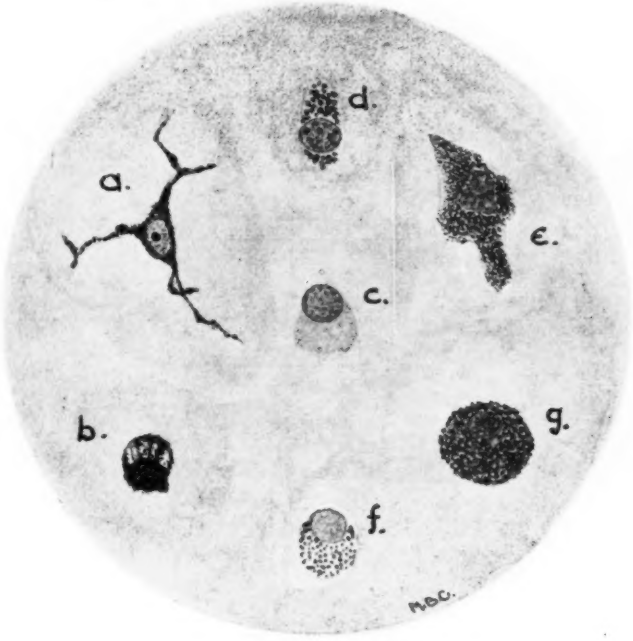

FIG. 1.—a. and b. are altered microglia cells; c.-g., granule cells; c., stained by toluidin blue; d.-f., contain granules of lipochrome, melanin and iron-containing pigments; g., a typical 'fat granule cell'. (× 1,000).

lymphocyte, but their cytoplasm, which is abundant and stains very feebly, is often only delimited by the granular content of the cell. This granular material within the cytoplasm of these cells appears to be part of the product of disintegration of the nerve-cell cytoplasm, which is transported by them through the tissues to be deposited in the lymph spaces surrounding the blood-vessels. This is shown by the fact that substances present in the healthy nerve-cells gradually disappear from their cytoplasm during degeneration and

are found instead in the perineuronal satellites, in the granule-cells wandering in the tissues, in those lying along the blood-vessels and in the perivascular spaces of these vessels, whereas in healthier areas granules of such material are not found in the satellite cells or perivascular spaces. Much of this granular material is of indefinite nature, the particles being recognisable only by reason of their affinity for some aniline dye or because they become deeply impregnated in silver preparations, but a number of fairly well-defined substances are also present in abundance. Thus, wherever nerve-cells containing much

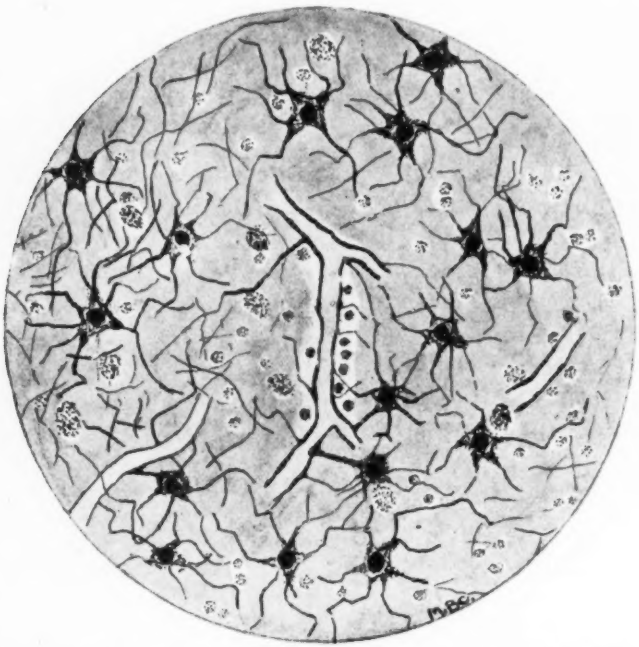

FIG. 2.—Showing the situation of the granule-cells in relation to a small capillary; they lie outside the membrana limitans perivascularis. Prefrontal cortex; Case III. Cajal's gold-sublimate method. (× 275).

lipochrome are degenerating, granules of this yellow pigment are readily recognisable in the granule-cells; many of the granule-cells in the substantia nigra are packed with fine granules of melanin; a few of these cells in the basal region contain granules which give a well-marked Prussian-blue reaction. In these cases the ordinary fat granule-cell was found in small numbers only in the region of the substantia nigra, where there had been a considerable amount of myelin sheath destruction. (Fig. 1).

The perineuronal satellites* may lie at a little distance from the nerve-cell but are usually situated very close to it, frequently in little bays at its periphery but never entirely within its cytoplasm. The granule-cells in relation to the precapillary and capillary vessels lie in the nervous tissues, between the vascular foot-processes of the astrocytes; they are closely applied to the glial membrana limitans perivascularis but lie outside it, and not in the perivascular spaces as the expression 'perivascular round-cell infiltration' would imply. (Fig. 2).

(In describing the pathological appearances in individual cases, figures

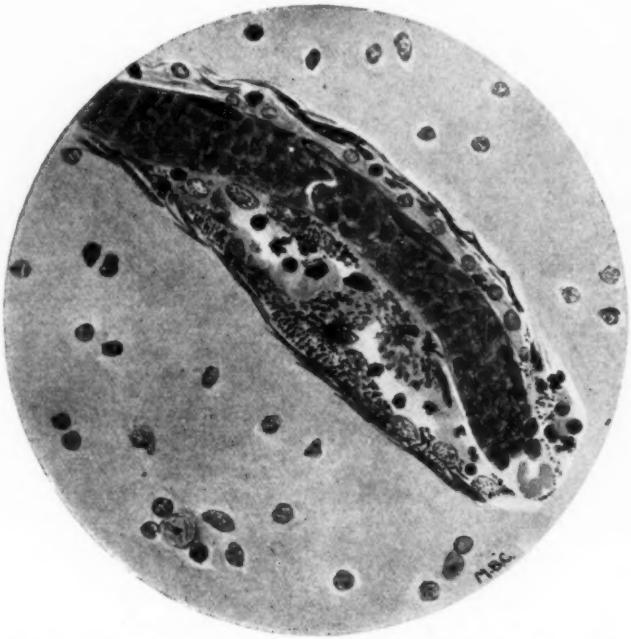

FIG. 3.—Disintegration of small granule-cells within the lymph space of a small subcortical artery. Prefrontal cortex: Case III. Haematoxylin-eosin-safran. (× 500).

will frequently be given indicating the numbers of these small cells in the groups about the nerve-cells and in the rows along the precapillary vessels, since this probably gives the clearest indication of the intensity of the reactive processes in different areas.)

In areas in which active changes have evidently been in progress for some time, very degenerate granule-cells with small, deeply-staining, gnarled,

* This expression will, in general, be used in referring to the small cells clustered about the nerve-cell bodies since, in routine preparations, the abnormal elements cannot usually be distinguished from the normal microglial and oligodendroglial 'satellites' of the neurones.

pyknotic nuclei may be found in the adventitial spaces of the larger vessels and can, very rarely, be seen penetrating the adventitia to reach the lymph spaces, in which they disintegrate. (Fig. 3).

Since most of the material had been fixed in bulk, extensive investigation by the methods of Rio Hortega could not be carried out, but sufficiently good preparations were obtained to suggest that the granule-cells in these cases arise from the microglia.†

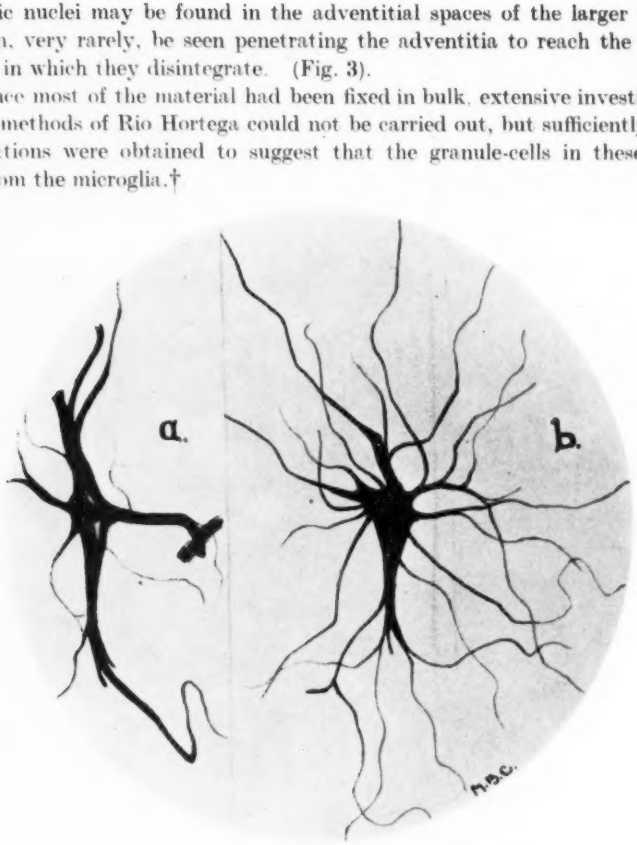

FIG. 4.—Large astrocytes with thick, fleshy, branching processes in which fibres are becoming differentiated. a., from the nucleus of the spinal tract of the trigeminal: Case III. b., from the globus pallidus: Case II. Anderson's Victoria blue method. (× 500).

Astrocytes.—In certain areas in which nerve-cell degeneration and granule-cell reaction are well-marked, as in the deeper layers of the cortex, changes in the astrocytes are insignificant: there is only a slight hypertrophy of a small proportion of the cells. In parts of the brain-stem, on the other hand, where other alterations are not so striking, the astrocytes are increased in number and many of them are greatly enlarged and actively engaged in glia-fibre

† Excellent descriptions and illustrations of microglia will be found in the papers of Penfield[15,16] and of Reynolds and Slater[19].

production, while the very occasional presence of bi-nucleated cells indicates that slow proliferation is still occurring.

The earlier stages in the development of the hypertrophic fibrous astrocyte are not very frequently seen but occasionally, especially in the nucleus of the spinal tract of the trigeminal, large glia-cells are encountered in which the cytoplasm of the cell-body and long, thick, branching processes stains very deeply with the aniline dye but contains no fibres. (Fig. 4a.) At a somewhat later stage the cell-body and processes take the stain rather less intensely,

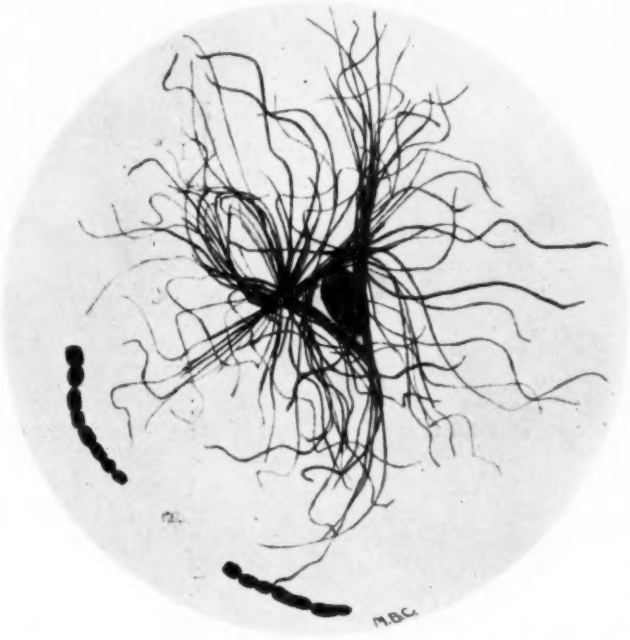

FIG. 5.—A hypertrophic astrocyte with voluminous bundles of well-formed fibres. From the substantia nigra: Case III. Anderson's Victoria blue method. (× 800).

while fibres make their appearance as deeply-staining condensations of the cytoplasm running along the edges of the processes and passing through the perinuclear zone from one process to another. (Fig. 4b.)

In the progressively altered cells most commonly met with, however, abundant fibre-formation has already taken place. The most striking examples of this type of cell are found in the substantia nigra of the Parkinsonian cases. The nucleus of these cells is usually large, oval or slightly irregular, with scanty

chromatin, arranged in a thin layer about the nuclear membrane, clumped into one or more nucleolus-like masses and scattered in fine granules throughout the karyoplasm. The cytoplasm, which stains very faintly with the aniline dye, is visible only in the voluminous cell-body and in the bases of the more massive protoplasmic processes. The long, stout, well-formed glia fibres, sweeping in graceful curves through the perinuclear zone, pass out through the larger processes in compact bundles or leave the cell-body singly or in little groups. In the larger processes several fibre bundles run along entwined together for a short distance and then turn off in different directions into the neighbouring tissues where they rapidly become resolved into spreading leashes of individual fibres, which mingle inextricably with those of adjacent astrocytes. (Fig. 5). At a later stage, particularly in areas in which much fibre-formation has taken place, the nuclei of the astrocytes are shrunken and the individuality of cells is lost in the dense fibre feltwork.

There can be no doubt that hypertrophy and proliferation of the glia-cells are a late component of the whole pathological process. Their absence in certain situations where other alterations are intense indicates this quite clearly. On the other hand such proliferation may be an active accompaniment of other changes, as in the nucleus of the spinal tract of the trigeminal, or may remain active in areas in which the granule-cell reaction is dying down and the majority of the surviving cells are healthy, as in the substantia nigra.

It seems probable that the astrocytic reaction is a purely reparative process, although it may vary, perhaps, in effectiveness in different situations or in different individuals. In certain little patches of grey matter in the thalamus which had been almost completely denuded of nerve-cells, gliosis was intense, whereas there was little alteration in the astrocytes in the surrounding nerve substance. The marked gliosis in the substantia nigra, also, was quite evidently related to an outfall of nerve-cells and fibres. But, of course, in a disease such as this, in which the grey matter is so diffusely affected, there may be obvious glial proliferation without any apparent outfall of specific nervous structures, and therefore the possibility that a late stimulating effect of the irritant itself may influence the astrocytic reaction cannot be wholly excluded.

Blood-Vessels.—Changes in the walls of the blood-vessels in these cases are confined to the region of the adventitial (Virchow-Robin) spaces and are largely, if not entirely, secondary to the alterations in the nervous tissues proper.

When numerous granule-cells are collected about the precapillary vessels the adventitial cells of these channels invariably show slight swelling and contain granular fragments and irregular masses of debris. This debris, which corresponds in character with the material found in the cytoplasm of the granule-cells and derived by them, in turn, from the degenerating nervous elements, includes fats, pigments and various other substances. Presumably these pass from the granule-cells lying in close apposition to the membrana limitans perivascularis through that membrane into the cytoplasm of the

adventitial cells. As already mentioned, a few degenerate granule-cells may be found in the lymph spaces of the larger vessels. In the substantia nigra there is usually well-marked perivascular infiltration, the infiltrate consisting of large and medium-sized mononuclear cells together with a large number of small cells. The nature of these small cells cannot always be determined when the infiltration is dense; some of them are probably granule-cells and the remainder lymphocytes.

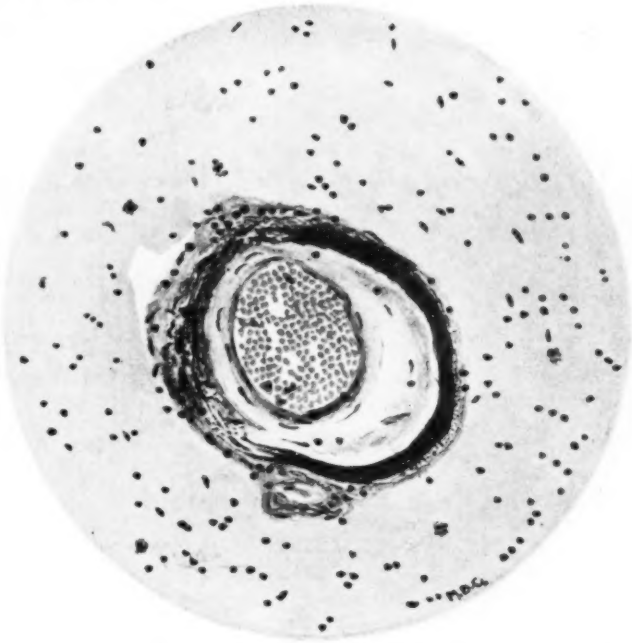

FIG. 6.—Large deposits of an iron-containing pigment in the media of a medium-sized artery; marked hyaline thickening of the intima. Globus pallidus; Case II. Hæmatoxylin-eosin-safran. (× 200).

Subsidiary Features. Hæmorrhages.—In the pia-arachnoid over the hemispheres, brain-stem and spinal cord small areas of hæmorrhage are often noted in the meningeal meshes close to the nervous tissues. In the cerebral cortex, where such extravasations are especially common, blood is frequently found tracking along outside the wall of a small cortical vessel to form a little pool beneath the pia or along its adventitial spaces to escape into the subarachnoid space. In the latter situation there is usually a great collection of larger and smaller mononuclear cells around the area of hæmorrhage, there is active phagocytosis of the remnants of the red cells and the cytoplasm of the macro-

phages contains granules of hæmatogenous pigment. Since there is never any indication of repair of the tissues following such hæmorrhages during the course of the illness it is concluded that they are merely terminal.

Vascular 'Siderosis,' etc.—Impregnation of the media of the arteries in the anterior half of the globus pallidus by an iron-containing pigment was noted in all four cases. In the first two cases described the pigmentary deposit was copious throughout the muscular layer of the vessel walls and there was usually a partial impregnation of the internal elastic lamina and of the region of the adventitial lymph spaces. A large proportion of the vessels were affected and, when marked, the condition was associated with great hyaline thickening of the intima, the lumen of the vessel being frequently reduced to less than half its original calibre. (Fig. 6). In the other cases the vessels showed only a partial impregnation of the media.

A number of 'degeneration bodies' were found in the region of the basal nuclei. Most of these were irregular, lobulated masses of large size (15-50 micra in diameter) which stained very deeply with hæmatoxylin and usually gave a well-marked Prussian-blue reaction. Smaller spherical granules were occasionally found near the capillaries.

These concretions are described in detail by McAlpine[11], Hurst[8] and others who have studied them extensively along with the arterial lesions. According to Hadfield[4], the latter are found in at least 60 per cent. of people over the age of 30, but are uncommon in younger individuals. The only remarkable feature here, therefore, is the great severity of the condition in a child of 13. (Case II).

HISTOLOGICAL FINDINGS IN THE INDIVIDUAL CASES.

CASE I.

Clinical History.—A female, aged 37, was admitted to the Royal Infirmary, Edinburgh, under the care of Professor W. T. Ritchie, in June, 1924; she complained of lethargy, insomnia and nocturnal restlessness which had persisted since a typical lethargic attack of epidemic encephalitis two months previously.

She had the characteristic Parkinsonian facies, attitude and gait; her voice was weak and toneless and her articulation slow; there was slight enfeeblement of the muscles of the left side of the face, affecting especially the left angle of the mouth, but no paresis of the limbs was noted and no abnormal movements were present.

She was finally discharged but was readmitted for a period of three weeks, some ten months later. By that time the signs of Parkinsonism had become more pronounced, salivation was constantly excessive, and the patient had become depressed and emotionally unstable.

In June, 1925, she entered the Royal Edinburgh Hospital for Mental Disorders as a voluntary patient. She was dull and depressed, apathetic and drowsy, and lay in bed almost stuporose, paying no attention to anything and often refusing to be fed; yet she was sometimes capable of conversing quite rationally with her husband.

Eventually she developed a septic broncho-pneumonia and died on December 28, 1925.

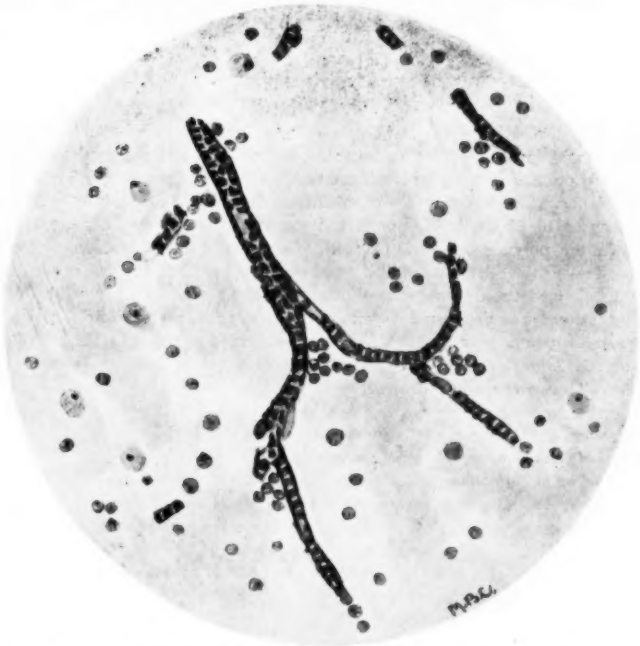

FIG. 7.—A group of small granule-cells in the fork of a precapillary vessel. Layer of medium-sized pyramidal cells; precentral cortex; Case I. Hæmatoxylin-eosin-safran. (×75).

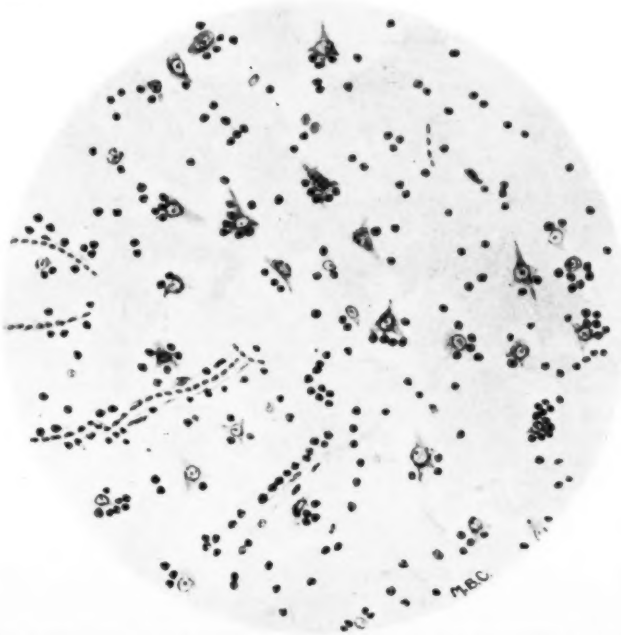

FIG. 8.—Cell degeneration and neuronophagia in the layer of polymorphic cells; prefrontal cortex; Case I. Toluidin blue. (×200).

Histological Findings.—Cerebral Cortex.—Beginning in patches in the layer of medium-sized pyramidal cells, severe degenerative alterations in the nerve-cells became increasingly prominent at deeper levels and reached their maximum intensity in the deepest cortical zone. Pericellular groups of satellites commonly numbered 6–8, and occasionally as many as 10–14 small cells were found clustered about a very degenerate nerve-cell deep in the polymorph layer. (Fig. 8). Large numbers of small granule-cells were grouped about the dilated precapillary and capillary vessels in the deeper cortex, rows of 12–15 being commonly encountered alongside these channels. (Fig. 7). A number of the astrocytes in the deeper cortex and adjacent white matter were slightly enlarged. Degenerate granule-cells were present in considerable numbers in the nervous tissues around the larger subcortical vessels but were rarely found within their perivascular spaces. There were numerous small areas of hæmorrhage in the pial meshes.

The changes outlined above were rather more marked in the prefrontal and temporal cortex than in the region of the fissure of Rolando, while in the occipital region only patches of the deeper cortex were severely affected.

Basal Region. Putamen and Caudate Nucleus.—Most of the small polygonal cells in these nuclei were very degenerate but the large multipolar cells generally showed only mild central chromatolysis. Neuronophagia was marked and as many as 14–20 satellites were occasionally found clustered about a disintegrating nerve-cell. Along the precapillary arterioles and venules there were frequently continuous rows of 10–15 granule cells. Many of the astrocytes were slightly enlarged.

Claustrum.—The changes resembled those in the putamen but were more severe.

Globus Pallidus.—Most of the large pallidal cells were fairly healthy and satellites were present only in relation to rare degenerate cells. Many of the astrocytes were slightly hypertrophic.

Thalamus.—There were a number of small foci of intense gliosis in the grey matter and in these foci the nerve-cells were conspicuously reduced in number. Elsewhere the thalamus was affected to about the same extent as the globus pallidus.

Within the perivascular spaces of the larger vessels in the basal region of the brain there were a few degenerate granule-cells and mononuclear phagocytes and a large amount of lipochrome pigment.

Midbrain.—There were alterations of varying degrees of intensity in many of the grey masses at this level. Severe nerve-cell degeneration was most frequent in the colliculi and reticular substance and only slightly less marked in the red nucleus and central grey matter. Collections of satellites about the damaged cells frequently numbered 5–7 or more, and there was a slight diffuse increase in the number of small cells along the adjacent capillaries. The large nerve-cells of the oculomotor nucleus and of the mesencephalic nucleus of the trigeminal were uniformly healthy.

In place of the normal large cell-islets of the substantia nigra only a few scattered pigmented cells remained. The majority of the surviving elements were perfectly healthy, however, and satellites were present only in relation to a few damaged cells. Large hypertrophic fibrous astrocytes were numerous in the substantia nigra and central grey matter, and the glia-fibre meshwork in these regions was abnormally dense. In the other grey masses of the midbrain area hypertrophic fibrous glia-cells were sparsely distributed.

Many of the larger vessels in the midbrain showed slight fibrous thickening of the outer adventitia and some swelling of the adventitial cells. In the region of the substantia nigra the dilated perivascular spaces contained a number of small degenerate cells, a few large mononuclear phagocytes and a large amount of melanin pigment.

Pons and Medulla.—Severe degenerative changes, accompanied by an increase in the number of perineuronal satellites, sometimes up to 7 or 8, were frequent in the cells of the nuclei of the spinal tract of the trigeminal and tractus solitarius and in the reticular

substance. Occasional degenerate or atrophic nerve-cells were found in many other nuclei, but the large multipolar cells of the somatic motor nuclei of the lower cranial nerves were uniformly healthy. There was no definite excess of satellites along the capillaries. Only a small number of hypertrophic fibrous astrocytes were found in the grey matter.

Cerebellum.—Showed no pathological changes of importance.

CASE II.

Clinical History.—A female child, aged 13, was admitted to Edinburgh Royal Infirmary, under the care of Professor Murray Lyon, in September, 1926. Her complaint was that, since an attack of 'influenza' in February, 1926, she had become progressively thinner and weaker and had developed occasional backward twitchings of the head. She was found to be extremely emaciated and so weak that she could not sit up or feed herself without shaking all over, but no definite signs of organic disease were elicited. Moro's test and the Wassermann reaction were negative. On lumbar puncture, a clear fluid, containing a few lymphocytes but no organisms, came out under pressure. On October 24 she developed severe myoclonic movements of the face, arms and legs; five days later a spastic paralysis of the left side appeared; still later dysphagia and complete anarthria developed in turn and she became completely comatose on November 3, the day before her death.

Histological Findings. Cerebral Cortex.—In the superficial half of the cortex the nerve-cells showed only mild chronic changes, but from the level of the medium pyramids downwards severe degenerative alterations in the cells became increasingly prominent. As a rule perineuronal accumulations of satellites numbered only 3–4, but groups of 8–9 or more were quite commonly found around the deeper polymorphs. About the precapillary arterioles and venules little rows of 6–8 granule-cells in single layers, or groups of 9–10 where the vessel bifurcated, usually represented a maximum accumulation. A few of the astrocytes in the deeper cortex were slightly enlarged. Granule-cells were present in large numbers in the nervous tissues around the larger subcortical vessels and were occasionally found within their perivascular spaces. There were a few small areas of hæmorrhage in the arachnoid meshes and in the superficial cortex beneath the pia.

The pathological changes in the cortex of this brain were less extensive than in the other three cases, only patches of the deeper layers being severely damaged. Such patches usually shaded off through a zone of less intense reaction into tissue which was little affected.

Basal Region. Putamen and Caudate Nucleus.—There was widespread degeneration of nerve-cells throughout these grey masses, the small polygonal cells being much more severely damaged than the large pallidal cells. Around the degenerate nerve-cells there were groups of 5–6 or more satellites and along the small vessels rows of up to 10–12 granule-cells. A few of the astrocytes were slightly enlarged.

Claustrum.—The changes here resembled those in the putamen.

Globus Pallidus.—Only a few of the large pallidal cells were degenerate; there was little neuronophagia and only a slight increase in the number of satellites along the capillaries.

Thalamus.—Many of the nerve-cells showed degenerative alterations of moderate intensity. Accumulations of 6–9 satellites were found about some of the more degenerate cells and there were occasional rows of 5–7 granule-cells along the precapillary vessels. Many of the astrocytes in the thalamus were enlarged and a few hypertrophic fibrous cells of medium size were found here and there near the larger vessels. There was marked hypertrophy and proliferation of fibrous astrocytes in a few tiny foci which had become almost entirely denuded of nerve cells.

A few small pyknotic nuclei were found in the adventitial spaces of many of the larger vessels in the basal region of the brain.

FIG. 9.—A small group of healthy surviving cells in the substantia nigra. Case II. Toluidin blue. (× 275).

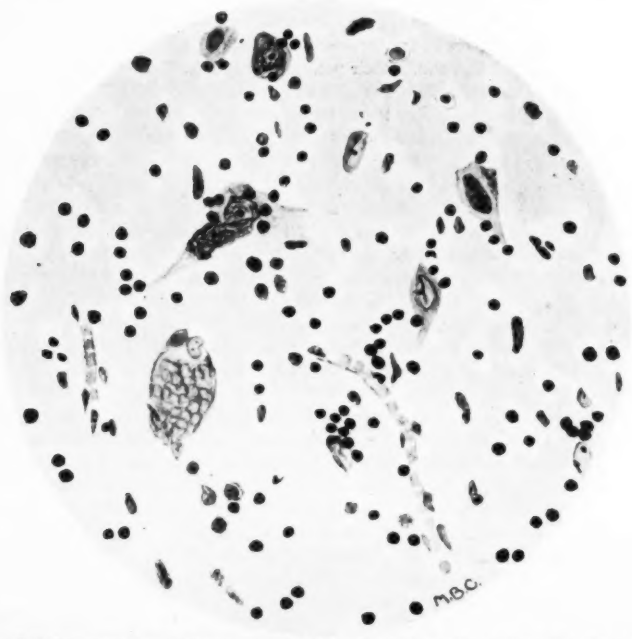

FIG. 10.—Cell degeneration and neuronophagia in the red nucleus. Case II. Toluidin blue. (× 275).

Midbrain and Hypothalamic Region.—In the colliculi, red nucleus, central grey matter and reticular substance nerve-cell degeneration was both frequent and severe and pericellular satellites were greatly increased in number, as many as 8–10 being frequently found in relation to the smaller nerve-cells. (Fig. 10). The large multipolar cells of the nuclei of the third and fourth cranial nerves, the mesencephalic nucleus of the trigeminal, the locus coeruleus, nucleus hypothalamicus and lateral geniculate body had almost all escaped damage but a number of cells in the medial geniculate body and substantia innominata were degenerate. The substantia nigra had sustained great cell loss but was much less completely denuded of cells than in the Parkinsonian cases. The majority of the surviving cells, moreover, were remarkably healthy in appearance and satellites were present only in relation to a few degenerate cells. (Fig. 9).

Compact rows of 6–8 granule-cells were present in relation to the precapillary vessels only where other alterations were well-marked. Elsewhere there was merely a slight diffuse increase in the number of satellites about the small vessels.

There was a considerable amount of glial proliferation in the substantia nigra, central grey matter and reticular substance, most of the astrocytes being fibre-forming cells of medium size. Elsewhere the astrocytes commonly showed some enlargement but fibre-forming cells were only occasionally noted near the vessels.

In most areas the lumina of the Virchow-Robin spaces contained only a few degenerate granule-cells but in the substantia nigra there were many large and small mononuclear cells within the meshes of the adventitia.

Pons and Medulla.—Only a small proportion of the cells of the motor nuclei of the lower cranial nerves were degenerate but in the sensory nuclei and, in particular, in the nucleus of the spinal tract of the trigeminal, many of the nerve-cells were severely damaged. Neuronophagia was usually very slight and there was nowhere any considerable excess of satellites along the smaller vessels. Many granule-cells were present in the tissues around the larger vessels, however, and small cells were occasionally found within the perivascular spaces. Many of the astrocytes in the posterior column nuclei and in the nucleus of the spinal tract of the trigeminal showed marked progressive alterations; occasional hypertrophic glia-cells were found in other parts of the grey matter especially in the floor of the fourth ventricle.

The *cerebellum* and *spinal cord* showed no noteworthy changes.

CASE III.

Clinical History.—A male, aged 62, was admitted to the Royal Infirmary, Edinburgh, under the care of Professor Edwin Bramwell, on November 13, 1927. Three years before he had had a typical, acute, lethargic attack of epidemic encephalitis; since then, he stated, he had felt very listless and his friends had noticed that his facial expression had changed, that his movements had become very slow and that he held his arms rigid in walking.

Fourteen days before admission he started to become progressively weaker so that after a few days he had to remain in bed and still later he became so weak that he could not feed himself or read for long; he had also developed incontinence of urine and faeces.

On examination the patient appeared dull, drowsy and confused; his memory was somewhat impaired but he answered questions intelligently, although with an effort and in a slow, monotonous voice. His face was immobile; the right naso-labial fold was smoothed out and the mouth drawn over to the left; saliva dribbled from his lips. His eyelids drooped markedly and he could not move his eyes upwards and only slightly from side to side. The masseters were weak, the tongue deviated to the right and was tremulous. There was no obvious weakness of the limbs but the muscles of both upper and lower limbs were hypertonic and there was a marked tremor of the hands on movement. Bed-sores were present over the sacrum and right malar eminence.

He gradually became more drowsy, developed a hypostatic pneumonia and died on November 20, 1927.

Histological Findings. Cerebral Cortex.—Degenerative changes were present in the nerve-cells in all layers of the cortex but were invariably slight in the cells of the superficial half, whereas in the deeper layers a large proportion of the nerve-cells were severely damaged. Collections of satellites about the degenerate cells varied in number from 3 up to 9 or 10, and continuous rows of 6–10 small granule-cells, with groups of 10–12 where the vessel bifurcated, were found in relation to the precapillary arterioles and venules. A small proportion of the astrocytes in the deeper cortex and adjacent white matter showed slight hypertrophy.

There were many degenerate granule-cells in the tissues around the larger subcortical vessels and a few of these cells were seen in the perivascular lymph spaces. At the surface of the brain there were a few areas of subpial and meningeal hæmorrhage.

The changes outlined above were fairly constant in extent and severity in all the regions examined with the exception of the occipital cortex, in which only patches of the deeper layers had been severely implicated.

Basal Region. Putamen, Caudate Nucleus and Claustrum.—The nerve-cells in these nuclei showed marked degenerative changes, more severe in the small polygonal cells than in the large multipolar elements. Around the degenerate cells there were groups of up to 8–9 satellites and along the precapillary vessels rows of 7–8 cells with occasional groups of 20 or more in double and treble layers. Many of the astrocytes were slightly enlarged.

Globus Pallidus.—Most of the large multipolar cells of this nucleus showed only slight chromatolysis but in a few, degenerative changes were more severe and about these small groups of satellites were collected. There was marked hypertrophy of a very few of the astrocytes.

Thalamus.—Most of the nerve-cells showed degenerative changes of moderate severity and satellites were collected in groups of 5–6 about the more degenerate cells and in rows of 6–7 along the small vessels. Only a few of the astrocytes were enlarged.

The larger vessels in the basal region of the hemispheres displayed marked swelling of the adventitial cells in the region of the perivascular lymph spaces. The cytoplasm of these cells contained a small amount of lipochrome pigment and a few degenerate granule-cells were present in the lumina of the spaces.

Midbrain.—Degenerative changes of varying degrees of severity were present in many of the nerve-cells forming the various grey masses at this level. In the colliculi, reticular substance and central grey matter, where almost all the nerve-cells were implicated, perineuronal satellites usually numbered 3–5, but quite commonly collections of 8–10 were present about badly damaged cells. The alterations in the medial geniculate body and red nucleus were only slightly less marked. Many of the large cells in the oculomotor nucleus showed slight central chromatolysis but only a few of these cells were more severely damaged. The substantia nigra had sustained great cell loss, for only a few scattered cells occupied the site of this large nucleus. Most of the remaining cells appeared fairly healthy, however, and satellites were present only in relation to occasional degenerate elements. In the tissues there were little groups of granule-cells collected around masses of melanin pigment, the remains of dead pigmented nerve-cells. Suitable preparations showed a marked outfall of axis-cylinders and myelin sheaths, particularly in the medial part of the nucleus. In most of the midbrain nuclei the perivascular satellites were increased in number but they rarely formed long continuous chains. The astrocytes in the substantia nigra were considerably increased in number; most of the cells were of the hypertrophic fibrous type and they had produced voluminous

bundles of fibres. (Fig. 11). The feltwork of glial fibres was very dense in the medial part of the nucleus. A number of hypertrophic fibrous astrocytes of a somewhat smaller type were present in the central grey matter, colliculi and reticular substance.

In the substantia nigra the perivascular spaces of the larger vessels contained many larger and smaller mononuclear cells and large amounts of melanin pigment and fat. Elsewhere very few granule-cells were found within the adventitial channels, although they were usually plentiful in the surrounding tissues.

Pons.—The large cells forming the motor nuclei of the fifth, sixth and seventh cranial nerves were generally fairly healthy but a few showed degeneration and around these small numbers of satellites were grouped. Degenerative changes of great severity were common in the cells of the superior olivary nucleus and nucleus of the lateral leminscus,

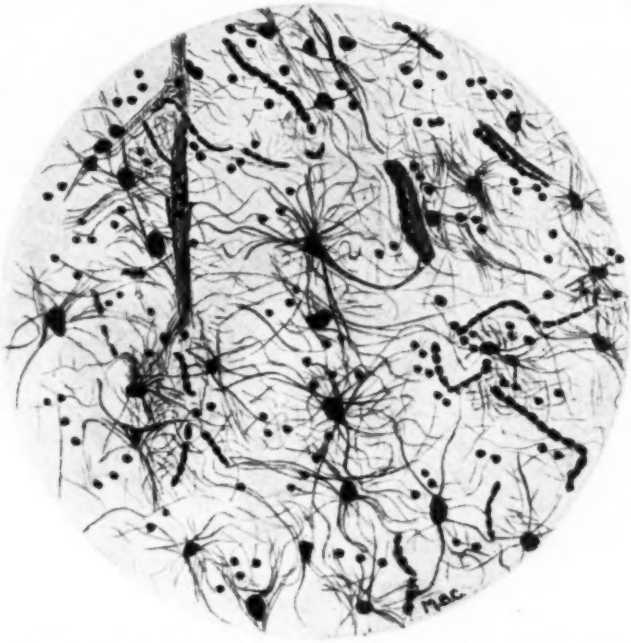

FIG. 11.—Gliosis in the substantia nigra. Case III. Anderson's Victoria blue method. (×275).

in the reticular nuclei and, in particular, in the main sensory and spinal nuclei of the trigeminal nerve. Five or six satellites were usually collected about the degenerate cells, but the satellites along the small vessels were only slightly increased in number. A number of hypertrophic fibrous astrocytes were scattered throughout the tissues.

Medulla.—The large multipolar cells of the hypoglossal nucleus, nucleus ambiguus and anterior column were almost all perfectly healthy. A number of cells in other groups showed mild degenerative changes but severe alterations were practically confined to the cells of the dorsal motor nucleus of the vagus, the nucleus of the tractus solitarius, the reticular nuclei and the nucleus of the spinal tract of the trigeminal, the last being most

extensively affected. Many hypertrophic fibrous astrocytes were found in this nucleus and in the grey matter in the floor of the fourth ventricle.

Cerebellum.—Showed no noteworthy alterations.

Spinal Cord.—Severe degeneration and neuronophagia were noted in a few of the nerve-cells in both anterior and posterior horns in the cervical enlargement and in many of the cells of the intermedio-lateral column in the midthoracic region. In this part of the grey matter the network of glial fibres was unusually dense; elsewhere only occasional fibrous astrocytes were found.

CASE IV.

Clinical History.—A female, aged 30, was admitted to the Royal Infirmary, Edinburgh, under the care of Dr. Chalmers Watson, in December, 1927. Following a slight

FIG. 12.—Marked accumulation of small granule-cells along a precapillary venule in the deeper layers of the prefrontal cortex. Case IV. Hæmatoxylin-eosin-safran. (× 200).

illness in January, 1927, she had become increasingly dull, listless, apathetic and irritable. No definite signs of organic disease of the central nervous system could be elicited. While in hospital she had occasional rigors during which the temperature rose to 105° F., but she stated that these did not cause her any discomfort. Numerous trophic sores developed during the few days preceding her death, which took place on December 19, 1927.

At autopsy no pathological changes in the organs were noted and there was no naked-eye indication of intracranial disease.

Histological Findings.—A slight degree of chromatolysis was apparent in almost all the nerve-cells throughout the central nervous system but this was regarded simply as the result of the fever and toxæmia accompanying the terminal illness.

Cerebral Cortex.—Nerve-cells showing severe degenerative changes were present in the second and third layers of the cortex but were much more numerous in the cortical zones at a deeper level. Neuronophagia was not usually very marked but groups of 8–10 pericellular satellites were not uncommon. Collections of 10–15 granule-cells, arranged in double or treble rows, were frequently found along the smaller vessels in the deeper cortex. (Fig. 12). A few of the deeper cortical and subcortical astrocytes were slightly hypertrophied.

Granule-cells were found in large numbers in the nervous tissues around the larger subcortical vessels but were rarely seen within the perivascular lymph spaces. In all areas there were many small hæmorrhages along the superficial cortical arterioles, at the surface of the brain beneath the pia, and in the meninges.

These alterations were all more intense in the prefrontal and temporal regions, particularly on the left side, than in the sensorimotor and occipital cortex.

Basal Region. Putamen and Caudate Nucleus.—Severe degenerative changes were present in the small polygonal and large multipolar cells throughout these nuclei. Groups of pericellular satellites usually numbered 3–12 and there were rows of 9–12 small granule-cells along many of the small arterioles and venules. Many of the astrocytes were slightly enlarged.

Globus Pallidus.—Many of the large pallidal cells showed moderately severe degenerative changes and had groups of up to 7–8 satellites around them. Along the precapillary vessels there were sparse, short chains of, at most, 4–5 granule-cells. Many of the astrocytes were enlarged and very rare large fibre-forming cells were encountered.

Claustrum and Thalamus.—The conditions in these nuclei resembled those in the globus pallidus but there were larger collections of satellites along the smaller vessels and no fibre-forming astrocytes were found.

The adventitial cells lining the lymph spaces of the larger vessels in the basal region of the brain were markedly swollen and within the lumen there were a number of degenerate granule-cells and a few macrophages laden with lipochrome pigment.

Midbrain.—A large proportion of the nerve-cells in the colliculi, red nucleus and central grey matter and a somewhat smaller number in the reticular substance and medial geniculate body showed severe degenerative changes. Groups of up to 9–10 satellites were collected about the damaged cells. Many of the large multipolar cells of the third and fourth cranial nerve nuclei were shrunken and atrophic. The substantia nigra of both sides showed a marked outfall of nerve-cells and fibres. Most of the remaining nerve-cells were shrunken and depigmented and there were groups of 5–6 satellites around a few very degenerate cells. There was a slight increase in the number of satellites along the small vessels throughout the midbrain area, always most marked where neuronophagia was most severe.

There was marked hypertrophy and proliferation of the astrocytes of the substantia nigra and a few large fibre-forming cells were also found in the central grey matter, colliculi and reticular substance.

Many granule-cells were found infiltrating the tissues around the larger vessels in the midbrain area but large numbers of small cells were found within the perivascular spaces only in the substantia nigra. Here the adventitial spaces also contained many large mononuclear cells and much melanin pigment and fat.

Pons and Medulla.—The large multipolar cells of the somatic motor nuclei of the lower cranial nerves were healthy. Severe degenerative changes were noted in the nerve-cells in the other grey masses in the lower part of the brain-stem but were frequent only in the nuclei pontis, in the main sensory and spinal nuclei of the trigeminal, in the nucleus of the tractus solitarius and in the reticular substance. Little groups of 5–6 satellites were usually present around the degenerate cells but there was no marked accumulation of

granule-cells along the precapillary vessels. A small number of hypertrophic fibrous astrocytes were scattered at wide intervals throughout the grey matter. They were most numerous in the nucleus of the spinal tract of the trigeminal and in the central grey matter in the closed portion of the medulla.

Cerebellum.—Showed no noteworthy alterations.

Spinal Cord.—A few scattered cells in both anterior and posterior columns in the cervical region showed severe degenerative changes; the mid-thoracic region had escaped with little damage; only a small group of cells in the middle of the anterior column in the lumbar region was implicated; in the sacral region a number of cells in the anterior column and a considerable proportion of those in the posterior column showed marked alterations. The satellite groups about the degenerate cells were large (9–10) in the sacral region, elsewhere they were much smaller. There was no definite increase in the number of satellites about the precapillary vessels and no marked alteration in the astrocytes at any level.

COMMENTARY.

When encephalitis became prevalent in Europe in 1917, it was quickly realised that the condition must be infective in origin. Two features, in particular, gave support to this conclusion: the disease showed a tendency to occur in small localised epidemics, and the cerebral lesions were definitely inflammatory in nature. But histological and experimental research, though vast in amount, has failed to place our knowledge of the etiology of the condition upon a substantial foundation. The precise nature of the causal agent of the infection remains obscure.

Attempts to explain the disease by attributing the lesions to organisms of well-defined morphological type have not been lacking. Rosenow and Jackson[20], for example, described a diplostreptococcus which occurred around the vessels in the diseased nervous tissues. Hilgermann, Lauxen and Shaw[8] reported the finding of protozoa, resembling the parasites of coast fever of cattle, in the blood, marrow, spleen, liver and ventricular fluid of fatal cases. These and other similar observations have not been confirmed extensively, however, and the great majority of workers believe that the causal agent of epidemic encephalitis belongs to the group of ultramicroscopic viruses.

But it has never been actually demonstrated that a filterable virus is the cause of the disease. Many experienced workers have made repeated attempts to infect animals with material from cases of human encephalitis and have failed to transmit an infection. The positive findings of other workers are inconclusive because they may be interpreted in different ways.

The earlier workers often relied upon the presence of histological lesions in the brains of inoculated rabbits as evidence of the transmission of the infection. This work is difficult to evaluate because the lesions described cannot always be distinguished from those of the spontaneous chronic encephalitis

of rabbits. This disease is characterised by the presence of meningeal, cortical, perivascular and subependymal collections of small round cells in the brains of affected animals. Only rarely does it betray its presence during life by producing cerebral symptoms. The condition is apparently rare in Great Britain, but is common in Sweden and in some parts of America. McCartney[13], working at the Rockefeller Institute, found that 55 per cent. of a group of 372 rabbits were affected. None of these animals had been injected intracerebrally. It is obvious, therefore, that unless it can be shown that the laboratory stock is free from this condition, the histological appearances in inoculated animals must be interpreted with great reserve.

During the Paris epidemic of 1919–20, Levaditi[10] and his fellow workers attempted to infect animals with cerebrospinal fluid, blood, brain emulsion and filtrates of the saliva and nasal mucus of a number of cases of the disease. A fixed virus was obtained by inoculating rabbits with brain emulsion from one case. The cerebral tissues of another case gave an attenuated virus. In their numerous other inoculation experiments the results were negative. The fixed virus, when inoculated intracranially or by various extracranial routes, produces a rapidly fatal acute encephalitis in the rabbit. This disease is identical, clinically and pathologically, with the encephalitis produced in the rabbit by inoculation with the herpes virus. And, moreover, it has been shown by cross immunity experiments that the virus itself is identical with the virus of herpes. Levaditi concludes, therefore, that the causal agent of epidemic encephalitis in man is the herpes virus or a special strain of this virus which has developed ' neurotropic ' tendencies.

Flexner and Amoss[2] have confirmed most of Levaditi's experimental findings, but question the validity of his conclusions. They inoculated animals with nasopharyngeal washings and brain emulsion from many cases of encephalitis with uniformly negative results. In addition, they tested a series of 100 specimens of cerebrospinal fluid (27 of which came from cases of encephalitis) for the presence of the herpes virus. These fluids were injected intracerebrally in rabbits and only one of them proved virulent. This specimen was obtained from a case of neurosyphilis which had been under observation for several years and had never shown any sign of encephalitis. These workers infer that the herpes virus may occasionally invade the fluids and tissues of the human body without producing pathogenic effects and that the occasional presence of this virus in the brain of human cases of encephalitis is quite accidental.

Further work, without pointing to a definite solution, has served to show how complex the problem is. On the whole, it may be said that it is still possible that the causal agent of epidemic encephalitis is a special strain of the herpes virus which has acquired a marked virulence for human nervous tissues. But it is more probable that the disease is due to a totally different virus which has never been transmitted to animals. All our definite informa-

tion about the pathogenic properties of this virus has been obtained from the study of the disease which it produces in the human subject.

An analysis of the pathological findings in the four chronic cases described brings to light several interesting features of the chronic stage of the malady.

(1) *The lesions are widespread in the central nervous system.*—In most of the work which has been carried out on the pathological anatomy of chronic encephalitis the main object has been the elucidation of anatomical and physiological problems. The aim of the observers was to determine the site of the lesion which caused the development of post-encephalitic Parkinsonism. The presence of marked destructive lesions in the substantia nigra in this disease was used as a basis for conclusions regarding the functions of this cell-mass and attempts were made to determine the course of the fibre-tracts connecting it with other nuclei. Naturally, attention was focussed upon the substantia nigra and less regard paid to other areas. McAlpine[12] and other authors quoted by him are in substantial agreement that the changes in the substantia nigra are of great importance. Lesions elsewhere in the brain-stem or basal nuclei if present, are generally regarded as insignificant. No important cortical alterations were found by any of these writers.

More recently, however, a few observers have reported single cases of chronic encephalitis in which the lesions were much more widespread. In some of these cases very marked cortical changes were found, in addition to the advanced lesions in the brain-stem. Chronic degenerative changes in the ganglion-cells of the cortex with perivascular infiltration and overgrowth of protoplasmic neuroglia were noted by Pjatnizky, Pawljutschenko and Michejew[17]. Zucker[22] described a case in which there was a marked outfall of nerve-cells with disturbance of the cellular architecture and marked proliferation of microglia and neuroglia in the cerebral cortex. He noted the absence of inflammatory changes. Holzer[7] and Meyer[14] described cases in children in whom the acute disease had been followed by changes in character. Curiously enough, although very marked lesions were found in the midbrain and hindbrain in both cases and in the thalami in one, neither of them showed any noteworthy alterations in the cerebral cortex.

The findings in the short series of cases described in this paper show that a widespread distribution of lesions in chronic encephalitis is by no means exceptional. In all of these cases the substantia nigra was most markedly affected, but lesions of varying degrees of severity were found in almost all the other nuclei of the brain-stem and in the basal nuclei and thalami. The spinal cord showed definite alterations in two cases and in all four the deeper layers of the cerebral cortex were markedly implicated. The cerebellum alone had escaped damage in every case.

It is clear that in chronic encephalitis the lesions are not confined to a limited area. Practically the whole of the central nervous system is affected.

(2) *The disease is active.*—From the pathological standpoint, the active nature of the lesions is the most important feature of the disease. Yet few writers on chronic encephalitis give much attention to this aspect of the subject. Zucker[22], however, pointed out that the lesions in the brain-stem and cerebral cortex of his case were by no means quiescent. Reynolds and Slater[19], in a recent paper, lay stress upon the progressive nature of the astrocytic reaction in the midbrain of a patient who died eight years after the primary acute attack.

In the four cases under discussion the lesions are undoubtedly active. This is equally true of every case, whatever the duration of the illness (longest —three years; shortest—nine months). The nerve-cells show degenerative alterations; granule-cells are assembled in large numbers about the nerve-cells and small blood-vessels; large fibre-forming astrocytes are present in many areas. These appearances are to be regarded as unmistakable evidence of an active pathological process. They offer a vivid contrast to the findings in the nervous system in old-standing cases of paralysis following poliomyelitis. Here a glial scar takes the place of nerve-cells which have been destroyed while the surrounding tissues are healthy. The lesions of ''chronic' poliomyelitis are healed lesions; the lesions of chronic encephalitis are actively progressive.

(3) *The grey matter of the central nervous system is affected diffusely but the lesions vary in intensity and in degree of development in different grey masses.*—The histological findings differ in different parts of the brain. But it is reasonable to assume that the underlying pathological process is essentially the same in all. It has simply advanced further in some regions than in others. Different nuclei, even when lying close to each other, may be very differently affected. The lesions may be severe in the substantia nigra, for example, and relatively slight in the adjacent red nucleus. Moreover, the appearances suggest that the process is increasing in intensity in some areas, while it is becoming more quiescent in others.

In every cell-mass affected, the lesions are diffusely distributed. In a given area, minor variations in the intensity of the reactive processes are frequently noted. But the disease is rarely definitely focal. A few small foci of intense gliosis, associated with a marked local outfall of nerve-cells, were found in the thalami of two cases. But elsewhere the disease always affected the tissues diffusely.

The variations in the intensity and in the stage of development of the pathological process in different areas may be illustrated by a comparison of the findings in the cerebral cortex with those in the substantia nigra.

Throughout the deeper layers of the cortex, degenerative changes in nerve-cells are frequent and severe, but there is little indication of actual cell-destruction; marked neuronophagia is common; granule-cells are accumulated about

the precapillary vessels in large numbers but are seldom found within the lymph spaces of the larger arteries and veins; the astrocytes show little alteration.

In the substantia nigra there has been a considerable outfall of nerve-cells and fibres; the remaining cells are frequently depigmented but, in the majority, the Nissl granules and neurofibrils appear fairly healthy; neuronophagia is seldom marked; the number of satellites along the smaller vessels is not excessive, but numerous degenerate granule-cells are present in the lymph spaces of the larger arteries and veins; the astrocytes are markedly increased in numbers and most of the cells are of the large fibre-forming type.

In the cortex there is a process of intense nerve-cell degeneration accompanied by a marked reaction of the phagocytic granule-cells, while the astrocytes remain practically unchanged.

In the substantia nigra this stage is almost over. Many of the nerve-cells have been destroyed; a few are degenerate; but the majority of those that remain have either escaped damage or have recovered. The astrocytes are engaged in the repair of the tissues.

These are the extremes. The conditions in other areas illustrate intermediate stages.

(4) *The pathological process is a relatively simple one, implicating only the nervous tissues proper.*—A study of the lesions at different stages of development enables us to obtain some idea of the sequence of changes occurring in the diseased nervous tissues.

Degeneration of nerve-cells may be noted in areas in which there is no neuronophagia, no increase in the number of satellites about the nerve-cells or along the blood-vessels and no proliferative changes in the astrocytes. But such cell degeneration is never absent when other changes are present. It must be regarded, therefore, as the primary alteration in the nervous tissues.

In most areas, however, the degenerating nerve-cells are surrounded by a number of small granule-cells, and there is a marked increase in the number of small cells along the adjacent precapillary and capillary vessels. These small granule-cells apparently arise from the microglia. They probably act simply as scavenger cells, absorbing the debris cast out of the disintegrating nerve-cell cytoplasm and depositing it in the adventitial spaces of the smaller vessels. Finally they degenerate and die, the nucleus becoming broken up or undergoing gradual lysis. This may occur in the nervous tissues, but very frequently the granule-cell passes into the lymph spaces of one of the larger vessels before disintegrating. Many of these cells are seen in this situation in areas in which an intense reaction has been in progress for some time.

The degenerative changes in the nerve-cells may become more and more severe and finally lead to the death of the cell. This is a frequent occurrence

amongst the large pigmented cells of the substantia nigra. But, in other areas, although degeneration of cells is common, complete destruction appears to be relatively slight. There is rarely any direct evidence of outfall of neurones.

When nerve-cells have been destroyed the astrocytes enlarge and may proliferate. They develop long, thick, branching cytoplasmic processes in which bundles of fibrils become differentiated. In this way a network of glia fibres is laid down and this repairs the gaps in the tissues resulting from the destruction of neurones.

This process involves essentially only the nervous tissues proper. Cellular elements derived from the bloodstream, from the walls of the blood-vessels or from the meninges, do not play any active part in it. The slight alterations in the region of the perivascular spaces are such as may result simply from the accumulation of debris in this situation. And this debris is brought to the lymph spaces by phagocytes originating in the nervous tissues themselves. These phagocytes were formerly termed 'gliogenic granule-cells' because they were believed to arise from the ectodermal astrocytes. Hence it was supposed that all the elements involved—the nerve-cells or their processes, which degenerate, the granule-cells which remove the debris, and the astrocytes which repair the tissues—were ectodermal in origin. The whole process was accordingly spoken of as an 'ectodermal reaction.' Within recent years, however, Rio Hortega, Penfield[16], Hurst[8] and others have demonstrated that the 'gliogenic granule-cells' arise, not from the astrocytes, but from the microglia. This is a special category of cells which is found only within the nervous tissues but is believed to be of mesodermal origin (Penfield[9]). The expression 'ectodermal reaction' can therefore no longer be regarded as accurate. But it may still be retained as a useful term indicating that the pathological processes are confined to the nervous tissues proper.

A pure 'ectodermal reaction' of this type follows injuries which inflict damage upon the nerve-cells or their processes without affecting the blood-vessels. When the vessels are injured, reactive processes of a totally different character are set in motion. These will be considered later in discussing the acute stage of the disease. In chronic encephalitis the perikarya of the neurones alone are severely affected. There is no evidence of direct damage to the axis-cylinders or their myelin sheaths.

The cause of this severe injury to the nerve-cells merits consideration. Toxic substances, finding their way into the nervous tissues from the circulating blood, might be expected to affect susceptible areas fairly uniformly. Any deficiency, in the circulating fluids, of substances necessary for the nutrition of the nerve-cells would likewise produce effects simultaneously in all areas. But, as has been pointed out already, the lesions in different areas vary greatly in severity. Moreover, in some cell-masses the disease processes are at an early stage of development, in others they have advanced far towards healing.

It would be difficult to attribute such lesions to injurious agents reaching the tissues through the circulating blood.

A great variety of local tissue alterations are capable of causing damage to the neighbouring nerve-cells. Local defects in the circulation of fluids, for example, resulting from disease of the walls of the blood-vessels, might cause local injury to the nerve-cells. But, in general, the walls of the vessels are healthy. Toxic substances might find their way into the adjacent nervous tissues from inflammatory foci in the walls of the vessels or elsewhere. But such inflammatory foci are absent. Living organisms could injure the nerve-cells very readily. But, if these are present, they cannot be demonstrated.

There is, in short, no evidence regarding any local or general factor, nutritional or toxic, to which the nerve-cell degeneration can be attributed. But it is reasonable to assume that the progressive lesions of the chronic stage of encephalitis are due to the action of the virus which caused the primary acute attack: that this virus lingers on in the tissues after the acute stage of the disease is over; and that it exerts a direct injurious effect upon the nerve-cells.

The severity of the lesions in the chronic stage of the disease may depend to some extent upon the particular strain of the virus which reaches the nervous tissues and upon its ability to survive and multiply there. It may depend upon varying degrees of susceptibility of the nerve-cells in different individuals. But it certainly does depend upon differences in susceptibility of different types of nerve-cell in the same brain, for some cell types are always much more severely affected than others. Many of the large pigmented cells of the substantia nigra are always destroyed, whereas there is no evidence of outfall of cells in the adjacent red nucleus. The small cells of the sensory nuclei of the cranial nerves are severely damaged, the large multipolar cells of the motor nuclei very slightly. And since these cell-masses lie very close to each other, it is obvious that the virus may reach any of them equally readily. The differences in the effects produced must, therefore, depend upon differences in the susceptibility of different types of nerve-cell.

A number of other problems still remain to be considered. Does the virus exert its action upon the nerve-cells slowly and continuously from the time it first gains entrance to the tissues? Or may it, in some cases or in some situations, remain for a time latent and practically inert, until circumstances favour the progress of the infection? In the case of the very chronic lesions in the midbrain region it seems impossible to choose between these alternative possibilities. The lesions in the cerebral cortex, in the basal ganglia and in some of the nuclei of the brain-stem, on the other hand, appear to be of fairly recent origin. If they had been in progress very long, some destruction of nerve-cells and some proliferation of astrocytes must almost certainly have resulted. Then how do such lesions arise? Are they caused by a fresh spread of the virus from some part of the brain-stem in which the disease has been progressing? Or are they due to the activation of a local infection that has

previously been latent ? The histological data do not provide us with any information bearing upon these questions.

COMPARISON OF ACUTE AND CHRONIC STAGES.

It has already been pointed out that in these cases of chronic encephalitis the lesions are concentrated in the nervous tissues. The blood-vessels are little affected by comparison. In the acute stage of the disease, on the other hand, inflammatory changes affecting the walls of the vessels are the most striking feature. Cellular elements derived from the tissues of the vessel wall and from the circulating blood become heaped up in the perivascular lymph spaces and emigrate into the adjacent tissues. Lymphocytes, larger and smaller mononuclear cells, endothelial and adventitial cells, mast-cells, plasma-cells and even polymorph leucocytes may all participate (Da Fano[1]).

This type of tissue alteration falls into the general group of ' mesodermal ' types of reaction, which are characterised by the activity of cellular elements derived from the connective tissue—vascular system. Such reactions are known to follow upon injury to the walls of the blood-vessels. Accordingly Guizetti[3] has suggested that the perivascular inflammatory changes in acute epidemic encephalitis are due to the " direct localisation of the germs of the infection on the walls of the veins."

There is, of course, a considerable amount of nerve-cell degeneration even in the acute stage of the disease, but this is usually a much less conspicuous feature than the alterations associated with the vessels. Indeed, it is generally agreed that damage to nerve-cells is usually not nearly so marked as the clinical features would lead one to expect (Hall[5]). And, moreover, much of the cell degeneration that does occur may well be due rather to intoxication and interference with the nutrition of the cells than to the direct action of the virus. This is suggested by the fact that recovery from the symptoms of the acute stage of the disease may be rapid and complete, whereas recovery from symptoms of the chronic stage either does not occur at all or is tedious and, usually, incomplete. And in the chronic stage, as has been pointed out, the degeneration of the nerve-cells is probably due to a direct attack of the virus upon the cells themselves.

As the disease passes into its chronic stages the alterations in the nervous tissues progress while the perivascular inflammation subsides. In this respect chronic encephalitis differs essentially from general paralysis of the insane, a disease which resembles the chronic stage of encephalitis in that the nerve-cells are attacked and destroyed by a virus which is present in the nervous tissues. The destructive effects upon the nerve-cells and the associated degeneration of fibres in general paralysis are, of course, much more extensive and severe. But the most striking difference between the two diseases is this : in general

paralysis the perivascular inflammatory condition persists, the lymph spaces of many of the vessels being crowded with proliferated endothelial cells, lymphocytes and plasma-cells, while in chronic encephalitis the ' mesodermal reaction ' in the walls of the vessels has died away.

From the foregoing it will be apparent that the acute and chronic stages of epidemic encephalitis differ markedly in their pathology as well as in their clinical features.

In the acute stage of the disease both the ectodermal and the mesodermal tissues of the brain are affected. The virus attacks the walls of the blood-vessels and stirs up a brisk reaction of the mesodermal type in the region of their adventitial lymph spaces. Such an inflammatory process must affect the adjacent nerve-cells by poisoning the fluids upon which they depend for their nourishment and by interfering with the circulation of these fluids. No doubt a number of cells are also injured by the direct action of the virus. But after a time the perivascular inflammatory reaction dies down. Probably a few of the nerve-cells have been destroyed but the remainder recover and resume their normal functions.

Whether recovery is permanent or not will now depend upon the susceptibility of the nervous tissues proper. If these tissues prove very resistant to the action of the virus there will be no chronic phase. If they are very susceptible, chronic symptoms may appear as a continuation of the acute disease. But usually there is an interval of apparently good health before the chronic disease develops clinically.

In the chronic stage of encephalitis the ectodermal tissues alone are affected. The symptoms are due to the direct action of the virus upon the nerve cells. The reactive processes which develop are of the ectodermal type. If the nerve-cells prove very susceptible the symptoms of the chronic stage will appear during the course of the acute attack or soon after it is over. If they are less vulnerable a long time may elapse before the damage is severe enough to cause any functional derangement. The virus may even remain latent for a time, doing little or no damage. Hence the possible interval of months or years between the acute attack and the appearance of symptoms of the chronic stage.

In many cases encephalitis develops as a chronic disease from the outset. There is no recognisable acute phase. In these cases, we may suppose, the virus does not provoke a widespread perivascular inflammatory reaction but enters the nervous tissues and slowly attacks the susceptible nerve-cells.

PROGNOSIS IN CHRONIC ENCEPHALITIS.

Certain of the data gathered during the course of this study have a bearing upon the question of the prognosis in chronic encephalitis.

It has been pointed out that the lesions are not simply the result of the primary acute attack of the disease but are progressive in nature. And although no dogmatic assertion can be made regarding the causation of these chronic progressive lesions, it is reasonable to assume that they are due to the prolonged action of the virus which was responsible for the primary acute attack. This virus, apparently acting directly upon the nerve-cells, may destroy them completely : or it may damage them in such a way as to interfere with their function without causing complete destruction. The prognosis will be favourable only if the progress of the infection can be permanently arrested. The chief aim of therapeutic measures should be the complete destruction of the virus. Attempts to relieve symptoms may only injure the nerve-cells and render them more liable to attack.

Even the complete elimination of the virus, however, will not ensure complete recovery. Nerve-cells which have been destroyed cannot be replaced either anatomically or functionally. In other organs destruction of cells is made good either by proliferation or by hypertrophy of surviving elements. But there is no evidence that nerve-cells can ever be replaced by regeneration or that surviving cells undergo hypertrophy. Even if regeneration did occur it would be useless unless the newly formed cells could re-establish the connections of those which had been destroyed and take their place in the formation of functioning nervous arcs. Similarly, hypertrophy of the surviving cells would probably not assist in the restoration of function.

The grouping together of nerve-cells in masses subserving a definite function has also an important bearing upon the results of disease in the central nervous system. The patient in whom the apex of a lung or the lower pole of a kidney has been destroyed by disease will suffer no inconvenience, provided the remainder of these organs is healthy. But the destruction of a small portion of the nervous system, containing a nucleus with important functions, will produce grave disabilities, however healthy the rest of the nervous tissues.

The great majority of workers (McAlpine[12] and others) agree that the lesions in the substantia nigra are the cause of post-encephalitic Parkinsonism. In every case in the present series there was a well-marked reduction in the number of cells in this nucleus. In every case the surviving cells appeared fairly healthy. Two of the cases, in which the outfall of cells was especially marked, had well-developed Parkinsonism. The other two cases, in which a much larger proportion of cells had escaped destruction, had not developed the Parkinsonian syndrome. It is apparent, therefore, that symptoms of Parkinsonism will not necessarily develop if a sufficient number of cells in the substantia nigra remain intact. But it is equally evident that once Parkinsonism develops, recovery is not to be expected since the nerve-cells in this nucleus are not simply damaged but are completely destroyed.

These remarks apply to the substantia nigra only. A very large number of other cell-groups were examined in the cases under discussion and lesions

were found in almost all. But in these a varying amount of nerve-cell degeneration was the rule, marked cell destruction the exception. Nowhere was there any evidence of an outfall of cells comparable with that in the substantia nigra. And, moreover, the damaged cells almost all appeared to be capable of recovery. There is reason to believe, therefore, that if the infection could be eliminated, recovery from symptom-complexes other than that of Parkinsonism would readily take place.

It is clear that in many cases of chronic encephalitis the virus remains active in the nervous tissues for years. Whether the infective process ever becomes arrested spontaneously is an open question. Many infections terminate naturally because the serum of the host develops substances or properties which are antagonistic to the parasite. It is possible that the serum of encephalitics may be capable of destroying the virus. But this would not necessarily affect the chronic disease, since it is probable that little, if any, fo the blood serum finds its way into the nervous tissues. The experiments of Teale and Embleton[21] show that a foreign serum, at any rate, does not penetrate into the nervous parenchyma. These workers injected guinea-pigs with large doses of horse serum and, after bleeding them thoroughly, tested their organs for the presence of this serum by delicate anaphylactic methods. It was found that the horse serum was present in the liver, spleen and omentum, but could not be detected in the brain or spinal cord. It is quite likely that the serum proteins of the animal itself are likewise excluded from the nervous tissues proper.

The adventitial spaces of the blood-vessels act as the lymph channels of the central nervous system. These spaces are in free communication with the subarachnoid space and drain into it. And since the cerebrospinal fluid normally contains only a small amount of protein (·018 per cent.), it is obvious that only a very small amount of serum protein passes from the interior of the vessels into the perivascular channels. This has an important bearing upon the progress of inflammatory conditions in the meninges and perivascular spaces because protective substances in the serum will reach the affected tissues in a state of high dilution. In bacterial meningitis the permeability of the vessel walls is altered during the course of the acute inflammatory reaction and a fluid rich in plasma proteins is exuded. But in epidemic encephalitis the protein concentration in the cerebrospinal fluid is normal or only slightly increased. Antibodies must therefore reach a high concentration in the blood serum before they can become effective in the dilute fluid of the perivascular spaces. It is possible that, in acute encephalitis, such effective concentration of antibodies is finally attained and the virus in the walls of the vessels destroyed. It is certain that the perivascular inflammatory reaction subsides. But the degenerative changes in the nervous tissues continue, possibly because the serum does not reach these tissues at all.

The important differences between the acute and chronic stages of the disease might be more readily explained on the supposition that a local cellular immunity developed in the tissues of the vascular adventitia but failed to develop in the nervous parenchyma. This may indeed be the correct explanation of the phenomena. It is obvious that all tissues must have some natural resistance to the action of irritants. But it is not certain that an increased resistance can be acquired by the tissues apart from the fluids in which they are bathed. In the present state of our knowledge, therefore, it appears better not to invoke theories of local immunity in suggesting a possible explanation of the findings.

No matter how immunity is produced, the virus, although still present in the brain, no longer injures the walls of the vessels or excites an inflammatory reaction in their adventitia. The inflammatory infiltrate of the acute stage is absorbed, the endothelial and adventitial cells regenerate and the vessels resume their former healthy state. But the disease processes in the nervous tissues continue, nerve cells are destroyed and, since regeneration of neurones cannot take place, complete restoration of these tissues is impossible.

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THE PROBLEM OF SOCIAL ADJUSTMENT.*

By

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The problems of adolescence are largely problems of adjustment to society and the success or failure of such adjustment will make or mar the whole subsequent life of the individual. Psychologically, success depends on the formation of an adequate organization of several instincts to form a sentiment of a social self which shall in large measure dominate the other sentiments in the personality.

McDougall defines a sentiment as the organization of emotional dispositions in relation to an object, and the formation of sentiments represents a relatively high level in the development of the personality. In the study of social adjustment, therefore, we have to consider what is the object of the sentiment, what are the particular emotional dispositions concerned in the evolution of the sentiment, and what are the guiding lines along which this evolution takes place.

First as to the object of the sentiment. This is not so simple as is that of many of the sentiments usually quoted as illustrations, for example, love or hate. In these the object is a distinct individual generally other than the self, though the self may be an object of a sentiment of love or hate, and a variety of emotional dispositions are organized in relation to that individual. Here, however, the object is more of an abstraction. Social adjustment depends on a duality, an adjustment between the self on one side and the world of humanity on the other. It cannot exist except in relation to both sides of this duality and therefore it is difficult to define the term 'social self' which has been used in connexion with this sentiment. We may consider the self from two aspects, the introverted self and the extraverted self. It may be that the expression 'self-regard' which McDougall uses and which seems to include the social self should only be used in respect of the first of these, for the introverted self is literally self-regarding. The concern is with the essential ego, its tastes, its interests and its impulses, but it is very difficult to get far in the definition of such an introverted self without becoming involved with the extraverted self, for the very expressions, tastes, interests and impulses imply something outside for which the individual has a taste or in which he takes an interest, or towards which he experiences an impulse; and when we come to consider such things outside, we are concerning ourselves with the

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extraverted self, nor can we go very far in a study of such objects extraneous to the self without coming up against extraneous individuals—other selves with which our own self interacts. It is then the self in relation to other selves which is the object of that sentiment which allows the individual to be an integral part of a community.

While at any moment any emotion primary or derived, simple or compound, may be in evidence in the relationship of ourself to others, the organization of the sentiment depends more particularly on certain of the recognized emotional dispositions or instincts. Of these the herd instinct is commonly supposed to be of prime importance, but we must realise that in human behaviour its nature has come to be very much modified, and we must beware of using the term ' herd instinct ' to explain human behaviour too glibly. In its primitive form the herd instinct involves a sense of unpleasure when the animal is in isolation and pleasure when in contact with his fellows, more especially in physical contact. The conative urge is therefore to seek such bodily association on all occasions. Allied to this instinct but distinct from it, each probably mutually facilitating the other, is that ill-defined and unnamed instinctive reaction which involves suggestibility, passive sympathy, and imitation.

With regard to the close physical contact such as the compact huddling together seen in the herd of bison under the influence of the gregarious instinct, we do not generally find that the human herd behaves in this particular way, unless the football crowd and the sands at Blackpool and other resorts on the first Monday in August are examples of this. Rather do we find the herd instinct expressed in the affective phase of unpleasure in loneliness and pleasure in the not too close presence of others, while for the rest it is represented by a general facilitation of suggestibility, sympathy, and imitation. As to the close physical contact, there is some indication that this has been so conditioned as to be carried over into the domain of sex, for in much of what is called sexual play—apart from the actual act of mating—this close physical contact plays an important part. Similarly, in parental relationships the same applies, so that this pattern of behaviour cannot be regarded as belonging solely to the herd instinct, at any rate as this instinct is manifested in human beings. It is to be noted that the herd instinct, both in respect of its affective phases and its facilitation of suggestibility, sympathy, and imitation is apparently less potent in the introvert than in the extravert, at any rate at conscious levels. This diminished potency, however, may be more apparent than real, for, as Jung has pointed out, what is lacking in consciousness often enjoys complementary importance at unconscious levels : we often find in the behaviour of the introvert ill-controlled and poorly differentiated flashes of what is generally regarded as typically extravert behaviour.

In man it is through his suggestibility, sympathy, and imitation that he manifests his gregariousness. He accepts the opinions of his compeers, he has a fellow-feeling for his kind, and his actions are too often stereotyped

imitations of those of others ; but it is through these reactions that he makes his social adjustments. It is a truism to state that education depends on this instinctive group to achieve its objects, and without it, it could achieve nothing at all. Yet, as we shall see, this instinct alone is not enough either for social adaptation or for education, for the mentally defective often exhibits all three phases of it in no small degree and yet is almost totally uneducable.

Before considering this point, however, we must refer to another instinct which has a strong influence in establishing the sentiment of the social self. I refer to the sex instinct. This powerful urge is not in itself concerned with the make-up of the sentiment : to describe social intercourse as a manifestation of sexuality is to my mind a mistake : but what sex does is to give a tremendous impetus to extraversion without which the true social sentiment cannot exist. With the development of sex the whole personality unfolds, the individual becomes intensely interested in a personality other than himself. Sentiments are formed so strongly organized that they are able to outweigh the demands of such a dynamically powerful instinct as self-preservation. Under the influence of these sentiments the individual may conquer fear, subdue anger, and still curiosity, and in this process of extraversion the developing social self is involved and takes advantage of it in the organization of its own sentiment.

We see then that the social sentiment requires these three instincts for its existence—the herd instinct to give it being ; suggestibility, sympathy, and imitation to give it material with which to build ; and sex to give it the drive towards extraversion ; but it also requires the integration of these and the emergence of something higher and unique in itself, before it can be recognized as the describable characteristic of the human race which we are discussing. Needless to say other emotional dispositions become involved in the sentiment and help to modify it or to round it off. Curiosity, fear, disgust, self-assertion and self-abasement are seldom absent from the developed sentiment, but they are not essential to it.

In the development of this sentiment physical agencies are important. Thus certain observations of Dr. Gesell on the effect of precocious puberty on mental development are of interest. Without going into details of the work of this and other observers, it is sufficient to say that intelligence is not affected either in normals or in mental defectives by precocity, but in social adjustments there is evidence of maturity greater than might be expected from the actual age of the subjects. This would indicate that the physical endocrine secretions of the sex glands as well as the sex instinct itself—if we can fully separate these—must be taken into account in the formation of the sentiment in question. This but serves to remind us that in any purely psychological discussion we cannot leave physiology very far behind.

If we wish to illustrate the fact that the emotional dispositions alone are not capable of forming the sentiment concerned with social adjustment, but that integration depending on higher levels of the nervous system is necessary,

we may study the efforts of the young child in this respect—or still better those of the mental defective, who presents a reasonably accurate slow-motion picture of human development, slow enough in many instances to allow us to see a static cross-section of the process. The fact that physical development, more particularly that of the sexual system, distorts this picture to some extent, does not invalidate it for our particular purpose, for in view of what has been said about the influence of the sex instinct in promoting extraversion and stimulating the development of the sentiment it becomes clear that this stimulus alone is not enough, but that the capacity for integration must also be present.

It is a truism that the young child is not a social animal, his behaviour being almost entirely egocentric, and that only gradually does he even recognize an increasing circle of individuals in his environment and still more slowly learns to adapt himself to them and to give and take in a proportionate manner. In the case of the defective, one of the most striking observations of the recent report of the departmental committee appointed to examine the subject of mental deficiency in Great Britain was, that important as was defect in intelligence, almost more important was the obvious failure in social adjustment.

Whether we are dealing with normals or defectives, we find that all individuals exhibit the instinctive bases referred to above in varying proportions, and that defectives show no particular peculiarities in this respect. It has been maintained by some that defectives are more suggestible than normals, by others that they are less so. Observations by several American observers show that so far as innate suggestibility is concerned, there is no special difference discoverable between large numbers of defectives and large numbers of normals; but when it comes to the control and discrimination of this innate disposition in the course of development, matters are very different. Normally, knowledge is integrated: suggestibility is controlled to a greater and greater extent as age advances by increased knowledge, by the organization of beliefs, and by the development of the power of making judgements. At the same time, a certain amount of discrimination becomes possible between good and bad suggestions. In all this the defective has a poor chance, for his knowledge is scanty, his beliefs uncertain, and his judgements at fault. Therefore his control and discrimination of suggestion, sympathy, and imitation are poor. Similarly, in respect of the sex impulse, there is no reason to suppose that defectives in general are necessarily more strongly sexed than normal, but they are unquestionably less well controlled; hence when a defective has a strong sex impulse, he or she is more likely to break out into behaviour which gives copy to the more sensational journalism.

It is then in the three functions of control, integration, and discrimination that the mental defective is conspicuously lacking, and without these, full social adaptation is impossible. These three functions are associated with the proper development of the cerebral cortex. This is amply proved by the advances in neurology which we owe to such men as Hughlings Jackson,

Sherrington and Head. Hughlings Jackson was the first to show that abolition of cortical function by disease or injury leads to what have been called release-phenomena, of which the epileptic convulsion is the typical example. This symptom illustrates the corollary that if the abolition is temporary the re-establishment of trans-cortical control leads to a disappearance of the symptom. Sherrington has demonstrated the integrative action of the nervous system and he and his followers have shown how, for example, in the field of motor phenomena the cortex is concerned with the integration of muscular contractions into purposive movements.

Head, in his study of sensory phenomena, has clearly shown that the cortex is concerned with the function of discrimination. The recognition of two separate points of contact of an æsthesiometer on the skin, the evaluation of shades of differences in heat and cold applications are impossible if the cortex is damaged, while the special senses of sight and hearing whose chief function is the discrimination of slight variations in the wave-lengths of light and sound are entirely dependent on cortical integrity.

These three functions then are all concerned in the major function of adaptation, and are essential to the proper development of social adjustment. They depend essentially on a fully developed cortex which only reaches fruition comparatively late in childhood. It is true that the infant is provided at birth with the full number of potential nerve-cells called neuroblasts, and he will never increase the number of these, but they are only rendered functionally active when they are transformed into neurones. This transformation is largely determined, as Sherrington has shown, by the proper stimuli which come through the distance-receptors—sight, hearing and to a less extent taste and smell—and represent the ' interests ' of the child. Failure of development of the full cortical activity and with this amongst other things the power of social adaptation, may thus arise from the following causes :—

An initial paucity of neuroblasts—primary amentia.

Failure of proper stimuli—amentia by deprivation such as occurs in the blind or deaf if compensatory training is not given.

Distortion of interests—as occurs in certain emotional repressions and dissociations, the mechanism of which has been the subject of much recent study and is still imperfectly understood, but which underlie the psycho-neuroses and probably some of the cases of the psychoses, and which may be ameliorated by analytic psychotherapy.

Social adjustment, then, must be regarded as of gradual development and only coming to fruition with the full functional activity of the cortex. Two pieces of work in which I have lately been associated with Dr. Thomas seem to illustrate this. It is sometimes supposed that even the higher grade mental defectives stop short in their development, advancing to a mental age of say six or seven, and then ceasing to make any further progress. In the serial examination of the intelligence of a number of feeble-minded children attending the special school at Bath, over a period of nine years, Dr. Thomas

found that far from there being any cessation of development, the mental age of these children, with only two temporary exceptions, continued to rise, slowly it is true, but none the less steadily ; and about the time of leaving the school, at 15 to 16 years of age, there was a decided steepening of the curve of development, showing a late spurt in the establishment of mental capacity. The theoretical normal increase is one year of mental age for every year of actual age, thus maintaining the intelligence quotient at 100 per cent. Since these higher grade defectives only increase their mental age about four to six months in every twelve months, the curve of the intelligence quotient drops, but this must not obscure the fact that these children do progress. This being so we come to the second series of observations. These were carried out on a hundred children referred from elementary schools as difficult in some respect or other. Almost all of these were found to be backward, but not legally defective. A critical study showed that apart from any intellectual defect which would of course make them difficult at school during the educational period, 50 per cent. had shown definite asocial tendencies of one sort or another during school age. Later, however, in the adolescent period, as many as 90 per cent. had made a reasonable adjustment in so far as they were in work and reported well of by their employers. This showed, therefore, that even after school age, which is commonly supposed to mark the term of mental development from the point of view of mental tests, progress in the power of social adjustments is possible. This was presumably attributable to further development of cortical function by the conversion of more neuroblasts into neurones, and by the provision of fresh stimuli and interests.

This brings us back to the subject of amentia. To the majority of people this implies defect in intellectual capacity and to suggest that a person considered by his neighbours to be highly intelligent was nevertheless an ament would probably be greeted with derision. None the less I make bold to assert that this is sometimes the case. Intelligence is not everything in life, for social adjustment is even more important ; and there are numbers of people who may or may not be intelligent who never develop the capacity of social adjustment. I suggest that many of these people fail to do so because they are lacking in the special cortical development necessary for the integration of their instincts and the formation of the sentiment which has been discussed.

People who lack this particular capacity seem to divide themselves into two groups. Firstly, there are those who compensate by an intense integration of an egocentric sentiment, so that there is nothing left over, so to speak, for social adjustment. It seems permissible to speak in this way because there is no reason to suppose that there is any centre for social adjustment in the sense that there is a visual centre or a centre for movements of the head and eyes. This function would seem to depend more on general cortical capacity than on the integrity of any localized anatomical area. In other words, this function, like many studied by Lashley, depends on the total number of neurones rather than on any special neurones. We are quite familiar with

individuals who exhibit certain 'capacities' apparently 'at the expense of' others, for example musical genius in the absence of general intelligence or the power of calculation and manipulation of figures in the absence of the power of making judgements and reasoning: thus there does seem sufficient evidence to warrant the assumption that in the absence of a sufficient total number of neurones all 'the eggs may be put in one basket.' Such people are conspicuously lacking in consideration for others and destitute of the capacity for affection, though they are sometimes apparently highly intelligent and may even be described as geniuses. Curiously enough this type of personality is in many cases associated with the illness of idiopathic epilepsy. Pierce Clarke and J. T. McCurdy in their study of epileptics have clearly recognised this asocial, egocentric personality and have called it the 'epileptic personality.' As has already been mentioned, the characteristic epileptic fit is a release-phenomenon and is a clear indication of failure in trans-cortical control. This conception of the epilepsies as being due to temporary or permanent deficiency in neurones is the only one which will include all varieties. The presumption is that the epileptic convulsion is due to a stimulus finding too ready a path for discharge. Sherrington has shown that the greater number of neurones a given stimulus has to traverse between the sensory end-organ and the final motor path the less violence will be exhibited in the motor response. If therefore a stimulus is short-circuited because there are not sufficient functioning neurones for it to traverse we might expect just the sort of thing that happens in an epileptic seizure. The stimulus may not traverse the higher neurones which normally control the motor field for several reasons:—

(i) Because the neurones are not and never have been there (amentia—idiopathic epilepsy).

(ii) Because the neurones have been destroyed (traumatic epilepsy).

(iii) Because the neurones have been temporarily or permanently put out of action by inflammation (encephalitic epilepsy).

(iv) Because the neurones have been temporarily or permanently put out of action by circulating toxins (toxæmic epilepsy).

(v) Because the neurones have been inhibited by emotional conflict (psychogenic epilepsy).

So whether we call epilepsy a deficiency disease with a restricted personality or whether we regard this particular variety of amentia, as I should prefer to do, as one which frequently exhibits, as an additional symptom of cortical deficiency, the epileptic fit, is a matter of little moment since the implication is the same.

The second group is not compensated and simply fails to adjust to life. Such are those whom Janet has called the psychasthenics and many of the chronic hypochondriacs who seem irrationally to prefer illness to health to the great annoyance of their friends and physicians: yet for these people ill-health with its attendant measure of protection and sympathy may be a

lesser evil, because much less difficult, than to have to stand on their own feet and adjust themselves to life. If this is so it is quite as unreasonable to expect such people to shake off their doldrums and undertake an independent existence as it is to expect the intellectually defective to understand the integral calculus, and analytic psychotherapy must not expect to be successful in 100 per cent. of its cases, for even this method cannot make up for a deficiency in neurones. I would like here to make it quite clear that I do not regard every neurotic as an ament. In the first place, adjustment to life is a relative matter, and the War showed that under great strain many people who would otherwise have continued to make a successful adjustment broke down. Further, what is strain for one is not so for another, so before we dub A an ament because he has broken down under strain X, let us be sure that strain Y would not prove us more of an ament than unhappy A. In the second place, we saw that deficiency in neuroblasts is not the only reason for failure in the development of cortical function, but that in many cases this is due to distorted interests. In such cases analytical psychotherapy may deliver the patient from his difficulties and enable him to make a successful adjustment quite up to the standard attained by the so-called normal.

I would conclude from this paper that :

(1) Social adjustment depends on the formation of a well-defined sentiment.

(2) The instinctive bases of this sentiment are herd instinct : suggestion, sympathy, and imitation ; and sex, together with other less constant contributory factors.

(3) These may be present, as in the mental defective, without the subject's having the power to achieve social adjustment owing to his deficiency in cortical tissue.

(4) The cortical functions of control, integration and discrimination are essential if social adjustment is to be achieved.

(5) The absence of social adjustment may be a sign of amentia apart from intellectual defect.

(6) Even in defectives this function of adjustment may go on developing in late adolescence ; hence an individual should not be regarded as hopelessly asocial too early in life.

(7) This absence of social adjustment may be associated with idiopathic epilepsy.

(8) Epilepsy is essentially a sign of some sort of cortical defect in function.

(9) The ' social ament ' may be apparently intelligent because one capacity has been developed at the expense of others.

(10) ' Social amentia ' may be part of a general psychasthenia which in certain cases is a real amentia, and therefore not amenable to psychotherapy.

THE VALUE OF ROUTINE EXAMINATION OF THE CEREBROSPINAL FLUID: REPORT ON THE EXAMINATION OF 3,200 FLUIDS.*

By

NEIL McDIARMID, Whittingham.

The examination of the cerebrospinal fluid is such a valuable aid to the diagnosis of nervous and mental diseases that it naturally forms a prominent feature of the work done in the laboratory of any mental hospital. A complete examination of the fluid is made in practically every case admitted to this hospital, and I have in this article analysed the results of examination of 3,200 fluids carried out in our laboratory during the last ten years.

In 2,400 normal specimens the fluid was clear and colourless, and there was no coagulum. The reaction was alkaline.

The protein content ranged between ·02 and ·035 per cent.: that of the chlorides between ·72 and ·75 per cent., the average being ·74 per cent. Fremont-Smith and Dailey[1] also regard the normal chloride content as from ·72 to ·75 per cent. The average cell-count was ·4 cells per c.mm., and ranged between 0 and 2·6 cells per c.mm., the cells consisting of large and small lymphocytes. The sugar content ranged between ·062 and ·089 per cent., the average being ·077 per cent.

Lange's gold sol test was constantly negative, as were also the colloidal gamboge test, the Wassermann reaction, and the Meinicke reaction.

I found no polymorphonuclear leucocytes in normal fluids, and this coincides with the results obtained by most recent workers. Fontecilla and Sepulveda[2], however, consider that normally cerebrospinal fluid contains 5 per cent. of polymorphonuclear leucocytes. Leredde, Rubinstein and Dronet[3] consider the number of cells in the normal fluid to range between ·5 and 1 per c.mm., and Levinson's[4] figures are 4 to 6 per c.mm., all the cells being small lymphocytes. Greenfield[5] gives ·3 cells per c.mm. as the normal finding.

Stowe[6], in an analysis of 556 normal fluids, found the average sugar content to be about ·075 per cent. Greenfield[5] gives the normal range as ·050 to ·075 per cent., while Levinson's[4] figures are ·064 to ·09 per cent. Alpers, Campbell and Prentiss[7] give the normal range as ·053 to ·084 per cent.

In dementia præcox, mania, melancholia, secondary dementia, acute confusional insanity, senile dementia and epilepsy, the fluid was found to be normal. Lochelongue[8] reported the fluid to be normal in epilepsy.

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General Paralysis of the Insane.—In 555 fluids examined before the commencement of treatment the following results were obtained.

The fluids were clear and invariably under tension. The protein content was constantly raised, and ranged between ·04 and ·06 per cent. The average sugar content was ·067 per cent., and ranged between ·055 and ·116 per cent. The chloride content ranged between ·72 and ·75 per cent.

A normal cell-count was found in 2·7 per cent. of cases, and the remainder showed a pleocytosis of varying degrees: in 66·6 per cent. it was between 2·7 and 50 cells per c.mm.: in 14 per cent. between 51 and 100 cells per c.mm.: in 14 per cent. between 101 and 200 cells per c.mm., and 2·7 per cent. gave a count over 200.

Small lymphocytes were the predominating cells; and the remainder consisted of large lymphocytes, 10 to 25 per cent.: 2 to 3 per cent. of polymorphonuclear leucocytes, and usually 2 to 5 per cent. of plasma-cells.

The Wassermann reaction of the fluid was positive in every case, and in only one case was the Wassermann reaction of the blood negative.

The Meinicke reaction was also positive in every case.

With Lange's gold sol test 95 per cent. of cases gave a so-called 'paretic' response such as 5554321000, 5554432110, or 4443211000. A better term than 'paretic' response or curve, for reasons which will be advanced later, is a 'zone 1' curve. The remaining cases gave a 'luetic' or 'zone 2' curve, such as 0014320000 and 1354321000.

The colloidal gamboge reaction showed a complete precipitation in the first three or more tubes in 95 per cent. of cases, typical readings being 222220 and 222210. A 'zone 2' curve, 111000, was present in 5 per cent. of cases.

The Braun Husler test was positive in 100 per cent.

Juvenile General Paresis.—The results coincided with those obtained in the acquired type.

The above results all refer to fluids withdrawn before the commencement of treatment by malaria or tryparsamide. Examination of the fluid following treatment by malaria, 235 cases of general paralysis having been so treated by us, revealed an improvement in all but three cases, but in no case was there a complete return to normal. The most common change was a decrease in the cell-count—this occurred in 90 per cent. of cases.

Protein content was lower than before treatment in 50 per cent.

The Wassermann reaction was found to be negative in 15 per cent. after treatment, while another 15 per cent. showed a diminution in the intensity of the reaction.

The colloidal gold curve showed an improvement in 30 per cent. of cases, and in 7·5 per cent. a 'zone 1' curve was changed to a 'zone 2' curve.

In 7 per cent. there was a combined improvement in protein, cell-count, Wassermann reaction and colloidal gold curve.

Excluding the gold sol, there was a combined improvement as regards protein, cell-count, and Wassermann reaction in 15 per cent.

The serological improvement or otherwise was found to bear no constant relation to the mental state of the patients. Some of the patients who showed very little serological improvement have been discharged recovered, while some cases with marked serological changes have exhibited little or no mental improvement.

TABES DORSALIS.—The fluid was clear and under tension and the cell-count was much the same as in general paralysis, as was also the type of cell present, but plasma-cells were rarely found. Protein was also increased in this disease. The average sugar content was ·07 per cent., while the chlorides were normal.

In 90 per cent. of the cases the colloidal gold test gave readings such as 0244310000, 0022100000, and 0012211000—typical luetic curves—no change occurring in the first one or two tubes and the maximum colour reaction in the fourth or fifth. Ten per cent. gave readings of 5555421000—typical paretic curves.

The colloidal gamboge test in 90 per cent. of cases gave readings of 111000.

The Wassermann reaction was positive in 85 per cent. of cases.

In one case the only abnormality in the fluid was a slight pleocytosis, while in another a positive Wassermann reaction represented the only change.

CEREBROSPINAL SYPHILIS.—The fluid was clear and under tension. The cell-count was mostly between 10 and 50 cells per c.mm., and consisted mainly of lymphocytes. Plasma-cells were never found in this disease. The protein content was increased to much the same extent as in tabes. The chloride content was normal, and sugar also showed little variation from the normal.

The Wassermann reaction was positive in 90 per cent. of cases. The colloidal gold curve was most frequently 0244310000 or 1344210000; but no fewer than 35 per cent. gave a 'zone 1' curve. In one case the fluid was completely normal.

Therefore, although general paralysis and tabes dorsalis are both types of parenchymatous syphilis, the results of examination of the fluid in tabes, it will be seen, approximated more to those found in the meningovascular type than to those found in general paralysis. In these two diseases the Wassermann reaction was generally weaker than in the last-named.

The term 'paretic curve' is a misnomer, because it is by no means pathognomonic of paresis. The Lange reaction is not constantly a 'paretic curve' in paresis, and a paretic curve is obtained in many cases which have the characteristic symptomatology of meningovascular syphilis and tabes dorsalis, with no symptoms of true paresis. It would, I think, be more satisfactory to use the term 'zone 1' curve instead of 'paretic' curve, and 'zone 2' curve instead of 'luetic' curve. Solomon[9] states that the presence of 'paretic' curves in almost 50 per cent. of cases of cerebrospinal syphilis shows that the presence of such curves does not differentiate cerebrospinal syphilis from paresis. They occur in multiple sclerosis, tabes, brain tumour, etc. Practically

identical conclusions have been reached by other workers in this field, notably Kafka[10] and Weigeldt[11].

It will be seen that, from the above results, it is impossible to differentiate between general paralysis, tabes dorsalis and cerebrospinal syphilis by means of the colloidal gold reaction. It has been held by various workers that malaria does not change a 'zone 1' curve to a 'zone 2' curve, but we have found this to occur in 7·5 per cent. of our cases treated by malaria.

Post-Encephalitis Lethargica.—In 22 fluids examined it was found to be clear in colour and under slight tension.

Fourteen fluids were completely normal. Four cases showed a weak luetic curve and of these, two had a sugar content of ·104 per cent. and ·102 per cent. respectively. In the latter case the cell-count was 295 per c.mm., and this was the only case which showed an increase in the protein content.

Four other cases showed a pleocytosis of 10, 12, 20 and 28 respectively. The sugar content, excepting the two cases above mentioned, ranged between ·062 and ·080 per cent.

The Wassermann reaction was normal in every case. Therefore, no constant findings occurred in this disease, and examination of the fluid is of little help as an aid to diagnosis in postencephalitic psychosis.

Acute Meningitis (meningococcal or pyogenic).—Twelve fluids were examined, and the colour varied from a grayish-white to a yellowish-green hue. In the initial stages it was turbid, but soon assumed the typical purulent appearance. Xanthochromia was present in some cases. Pressure was increased in all but one: it usually ranged between 300 and 900 c.mm. of water.

In every case there was a marked increase of protein.

The sugar content was reduced in every case, and the quantity present depended on the state of the disease. With the progress of the disease the sugar steadily diminished in every case: ·018 per cent. was the average reading at the height of the affection.

Chlorides were slightly reduced, the average being ·66 per cent.

All cases showed a marked pleocytosis, and in one it was as high as 7752 cells per c.mm. The differential count was 87·5 per cent. polymorphonuclear leucocytes, and 12·5 per cent. lymphocytes.

The colloidal gold reaction gave the typical meningitic curve, the greatest change in colour occurring in tubes 7, 8, 9 and 10. Slight variations occurred, but the most typical curve was 0000112342.

As the disease advances, the colloidal gold curve is more displaced to the right into the weaker concentrations, and if improvement occurs the curve is gradually displaced to the left into the stronger concentrations.

The following Table shows the changes which took place in the curve with the advancement of the disease in a fatal case. The gamboge reaction is given for comparison:—

	Colloidal Gold.	*Colloidal Gamboge.*
1st Puncture	0011221000	112000
2nd ,, ...	0000132210	012220
3rd ,, ...	0000112342	001220
4th ,, ...	0000012332	001220

TUBERCULAR MENINGITIS.—Ten fluids were examined, which were usually clear or slightly turbid. The average pressure was lower than in acute meningitis.

Protein was increased, but not to the same extent as in the acute type.

Cells were constantly increased and ranged between 200 and 1000 cells per c.mm. In most cases small lymphocytes predominated, but in one case polymorphonuclear leucocytes were in the majority. This may have been due to a mixed infection. Sugar was always diminished at the height of the disease, but in one case was normal in the initial stages.

Chlorides were constantly reduced, more so than in acute meningitis, the average being ·63 per cent. at the height of the disease. One case gave a reading of ·52 per cent., and such a low reading is considered to be pathognomonic of this condition.

The colloidal gold curve was in the meningitis zone, but slightly weaker than with acute meningitis, the usual curve being 0000123100.

CEREBRAL ABSCESS.—In four fluids out of five examined the fluid was clear: in the fifth it was slightly yellow.

Protein was increased.

The average sugar content was ·066 per cent.

The chloride content was normal, except in one fluid, which gave a reading of ·69 per cent.

The cell-count ranged between 6 and 756 cells per c.mm., but in only one case was it over 100. In all except the fluid with a count of 756, lymphocytes were the predominant type of cell. In the case mentioned polymorphs constituted 93 per cent., large lymphocytes 3 per cent., and small lymphocytes 3 per cent.

The colloidal gold curve varied from 0012100000 (practically normal) to a very weak meningitic curve.

TUBERCULAR COMPRESSION PARAPLEGIA.—The seven fluids examined showed the typical Froin's syndrome, which it is unnecessary to give in detail. In addition, the readings in the colloidal gold and gamboge tests were respectively:—

Colloidal Gold.	*Gamboge.*
1111221000	011220
1334455455	111220
0000111340	000120
5432100000	210000
0000111000	001100
0000112321	002210
0000011232	001110

DISSEMINATED SCLEROSIS.—Nine fluids from five cases were examined. The fluid was clear and the pressure normal in every case. One case had a cell-count of 15, but the others showed no pleocytosis. Sugar was normal except in one case, which gave a reading of ·124 per cent. Chlorides were normal.

The colloidal gold findings in four cases were 0122100000, 0012210000, 5444321000, 3320000000, while the fifth was completely normal.

CEREBRAL HÆMORRHAGE.—Six fluids were examined, of which three were slightly yellow, two yellow, one red and evenly mixed with blood. Protein was invariably increased. Sugar was slightly increased. A moderate pleocytosis was present in every case. The colloidal gold and gamboge in three cases gave respective curves as follows :—

Colloidal Gold.	*Colloidal Gamboge.*
1112220000	210000
0001210000	011100
0012210000	022220

OLD HEMIPLEGIA.—Nine fluids were examined and showed in every case a slight increase of protein. The average cell-count was 8·3, and the usual colloidal gold curve was 0022100000.

HERPES ZOSTER.—In 20 cases examined the most common abnormality was a mononuclear lymphocytosis, accompanied by a slight excess of protein, and this was present in 17 cases. In two of these the cell-count was over 50. One fluid was normal, while two showed excess of protein, without any accompanying pleocytosis. On further examination of each fluid about a month after the attack, it was found in three cases that there was a diminution but not a disappearance of the pleocytosis.

DIABETES MELLITUS.—The sugar content in five fluids was constantly increased, and ranged between ·256 and ·568 per cent. Diacetic acid and acetone were present in one fluid, and this was in a case of impending coma—otherwise the fluids were normal. There was no increase of cells, and no change in the colloidal gold reaction. The increase in the fluid sugar was parallel with that of the blood.

CEREBRAL TUMOUR.—Twelve fluids from six cases were examined. The fluid presented the following characteristics :—

(1) It was usually clear and colourless, but in two fluids was slightly xanthochromic, and the pressure was constantly raised.

(2) The cell-count was normal in two instances, in eight it ranged between 3 and 12·6 per c.mm., and in the other two the counts were 28 and 41 cells respectively. The cells consisted of large and small lymphocytes.

(3) Protein was increased in every case.

(4) Chlorides were normal.

(5) The sugar content was within normal limits in 10 fluids ; in two it was slightly raised.

(6) Two fluids showed no change in the colloidal gold, one gave a 'zone 1' curve, two showed a very slight change in tubes 4 and 5, and the remainder gave a meningitic curve 0000123100.

(7) The Wassermann reaction was negative.

Lange[12] in a report on a series of fluids from cases of cerebral tumour obtained the following results:—

(1) A typical gold curve with colour changes occurring in the dilutions above 1:80.

(2) A moderate increase of protein.

(3) Almost invariably a yellow coloration of the spinal fluid.

Mourez[13] analysed the fluids from eight cases of brain tumour and concluded that the most important findings in the spinal fluid were an increase in total protein with a low cell-count, and a characteristic gold curve. He also stated that xanthochromia when present was a most important sign.

Spurling and Maddock[14] have stated that the picture presented by the fluid in brain tumour is marked by a low cell-count, a high total protein value, a characteristic gold curve 0000123100, and no significant variation in the sugar content.

HUNTINGTON'S CHOREA.—Three fluids were normal, and Eskuschen[15] reported no characteristic changes in his examinations.

VON RECKLINGHAUSEN'S DISEASE.—The only abnormality in two cases was a slight pleocytosis, accompanied by a slight increase of protein. In another case the fluid was entirely negative.

JAUNDICE.—The fluid in two out of eight cases was yellow in colour, and the discoloration was due to urobilin.

PELLAGRA.—Three cases were examined, of which two were normal, and the other gave a cell-count of 34.

INFANTILE CEREBRAL PARESIS.—The five fluids examined were normal.

PERIPHERAL NEURITIS.—Four fluids were normal.

I am indebted to Dr. R. M. Clark, Medical Superintendent, for permission to publish the results of these examinations, which have all been carried out under his supervision.

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Editorial.

PSYCHOLOGY AND MEDICINE.

ATTEMPTS to explain certain phenomena in medicine by psychological conceptions are found far back in history, but with the advent of scientific method these attempts were shouldered off the field, because it was thought that psychology was unable to satisfy the requirements which science demanded. This objection was later removed, partly by a better understanding of the real nature of science, and partly by a reform in psychological procedure, which freed it from the alien influences which had long crippled its development and allowed it to conform whole-heartedly to the rules of scientific method. Psychological conceptions then began to be systematically applied to medical problems, and encouraging results were almost at once achieved. During the past half century the attack along these lines has been vigorously pushed forward, and in certain spheres of medicine, notably in the psychoneuroses, psychological conceptions have enabled us to attain a degree of understanding and of therapeutic control incomparably greater than that before possessed.

Psychology has therefore unquestionably succeeded in establishing a place for itself in medicine, but the validity of its methods, the limits of its sphere, and its relations to other modes of dealing with the problems of disorder are still subjects of dispute and confusion. The question is debated, for example, whether the psychoneuroses are essentially psychogenic or physiogenic, and there would seem to lie behind this the implication that one theory must necessarily be valid and the other invalid. The question in itself has a definite meaning, but the implication behind it is false and due to a confusion of thought.

The aim of science is to construct conceptions which will enable us to explain and to predict the sequences of observable phenomena. We are at liberty to construct these conceptions either in physical or psychological terms, provided in either case that the method of science is rigidly followed, and that the test of utility is satisfied. By utility is meant precisely that

capacity to explain and predict phenomena, or in other words to achieve command over our experience, which constitutes the aim of science.

Hence in such a sphere as the psychoneuroses the question of psychogenesis or physiogenesis can only mean an inquiry as to which method of attack is better adapted to explain the phenomena of the psychoneuroses, and the answer must depend upon the ability of conceptions couched in these two modes to satisfy the fundamental test of utility. Each can lay full claim to scientific validity, and it must be emphatically stated that the issue is not one of 'either—or' but of 'both—and,' save only that in so eminently practical a matter as therapeutics there must be a leaning towards that mode which is more profitable.

It can hardly be doubted that, so far as the psychoneuroses are concerned, the psychological method of approach is at present more fruitful. Since the latter years of the nineteenth century, when it was first consistently applied in the work of Janet, it has cast an increasing light upon the phenomena formerly called functional nervous disorder. The conceptions which it has built up have enabled us to obtain a considerable understanding of the genesis of those phenomena, and hence to devise methods of treatment which have achieved a large measure of success.

If this statement is justified the validity of the psychological attack is established beyond question. This does not mean, however, that an attack from other angles is thereby put out of court. The physiologist, for example, cannot be expected to admit that a whole section of medicine is to be placed outside his purview. On the contrary he is bound to endeavour to push his method into every field of action of the human organism. He must maintain that, given a sufficient increase of knowledge, he should be able to explain, in physiological and neurological terms, every sequence of human behaviour, not merely pathological events, but every process from the simplest reflex to the most complicated train of speculative thought. He may agree that so far he has not been able to contribute much understanding or practical help in the sphere of the psychoneuroses, but this can only be an incentive to further investigation, and not a proof of the unsuitability of his method.

Moreover the physiologist may legitimately claim that, in spite of his present lack of success, his method has great advantages over a psychological approach. It is more capable of objective verification, and its hypotheses can therefore be easily

proved or disproved in a way which psychology, with its multiplicity of divergent schools, has obviously failed to achieve. Again it can be integrated with chemical and physical methods of attack, and indeed with all scientific methods except the psychological and perhaps the biological. In other words it belongs to the group of the mechanistic sciences, and gains immense strength from the solidity of its companions, and the common aims which it possesses with them. Hence even a small advance in the understanding of the psychoneuroses achieved by a physiological method may be of great importance, because it means ground won for the group of mechanistic sciences, the group which constitutes the most efficient and productive weapon yet achieved by man.

It is therefore clear that the physiologist must be encouraged to pursue his researches into the phenomena of the psychoneuroses along every possible avenue. If he should meet with any considerable measure of success it is likely that the psychological approach, though not rendered in the least invalid, would be largely superseded, because of those advantages in the physiological method which have just been enumerated. In the meantime, however, the psychologist must push along his own road and claim the right to explain all he can.

It may be added that, although the physiologist cannot submit to any arbitrary limitation of his field, there is always the possibility that mechanistic science may ultimately prove to be applicable to only a portion of our experience. Certain phenomena, purposive action for example, show a resistance to inclusion within its structure, which may be insuperable, and it is worthy of note that in the psychoneuroses a factor akin to purposive action is clearly demonstrable. It is permissible to speculate, therefore, that some aspects at least of the psychoneuroses may finally be found to lie outside the field of mechanistic science. The psychologist may therefore comfort himself with the thought that, although the physiologist and after him the chemist and the physicist may take over much of the road which he has built, there may always remain a sphere permanently inaccessible to those other workers, but in which he can continue to add to our knowledge and power.

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[75] **Morphological study of the inferior olivary complexus in man** (Étude morphologique du complexe olivaire inférieur chez l'homme).—I. Bertrand and P. Mareschal. *Revue neurol.*, 1930, i, 705.

This is a very fine piece of anatomical work which does not lend itself to convenient summarisation and should be read in the original. It is based on the study of five cases and is accompanied by drawings and charts which fully explain the findings that are given.

J. V.

[76] **On the projection of the macula on the area striata in man** (Über die Projektion der Makula auf die Area striata des Menschen).—B. Brouwer. *Jour. f. Psychol. u. Neurol.*, 1930, xl, 147.

A case of thrombotic softening in the left occipital region is here studied. The lesion had caused for a period of years a right homonymous hemianopia with exclusion of central vision. The region damaged proved to be small and to be confined to the frontal part of the calcarine fissure. A retrograde degeneration was readily traced forwards to the external geniculate body, confirming views of the author based on experiments in animals. In the caudal half of the calcarine area lies the macular projection system; peripheral segments of the retina are represented more frontally. Whether the macular part extends further forwards could not be determined by this case.

S. A. K. W.

[77] **The principal sinistral types.**—C. Quinan. *Arch. of Neurol. and Psychiat.*, 1930, xxiv, 47.

Of 1,000 university students examined with reference to handedness and eyedness, 704 were right-handed and right-eyed; 185 were right-handed and left-eyed; 40 were left-handed and left-eyed; 36 were left-handed and right-

eyed: 35 are classified as ambi-laterals and other types. Therefore, 26·1 per cent of these young men are sinistrals.

Nearly the same percentage of sinistrals was found in a study of 317 Chinese school children.

There seems to be a marked tendency for sinistrals to concentrate in certain of the æsthetic vocations.

On the basis of data collected from 815 university students, it can be stated that sinistrals are definitely more musical in their tastes than dextrals.

In a survey of 693 neuropsychiatric patients, it was found that in dementia præcox and in most other forms of mental disease the percentage of sinistrals was 30·4, while in a mixed group of persons with constitutional psychopathic states the average value was 54·1 per cent.

It is suggested that sinistrals, especially those of the RL type, are apt to show signs of constitutional instability.

R. M. S.

NEUROPATHOLOGY.

[78] **The pathogenesis of hepato-lenticular degeneration** (Études sur la pathogenèse de la dégénérescence hépato-lenticulaire).—Max Schmidt. *Acta Psychiat. et Neurol.*, 1930, v, 163.

After a succinct description of the clinical features of several cases the writer notes that in two of these, acute in character, the changes in the liver were comparatively slight; in one which progressed for 14 years they were much more pronounced. He supposes that some toxic agent affects equally the hepatic parenchyma and the neuroglial network of the neostriatum: both classes of cell absorb the toxin, degenerate, and disappear.

S. A. K. W.

[79] **The tissue, cytological and vascular alterations of the central nervous system in the pathogenesis of cerebral hæmorrhage** (Le alterazioni tissulari, citologiche e vascolari del sistema nervoso centrale nella patogenesi dell' emorragia cerebrale).—G. Daddi. *Riv. di pat. nerv. e ment.*, 1928, xxxiv, 534.

In the cortices of subjects of cerebral hæmorrhage of all ages areas of alternating rarefaction and density occur round the vessels. This is specially noticeable in the white matter below the cortex.

In older people these are diffuse areas of chronic change which are independent of the hæmorrhage, but which may complicate the histological picture of the lesion. Astrocyte glial cells are the commonest feature in the lesions. The latter are of two kinds, one the usual accompaniment of chronic changes

of old age ; the others occur at any age and represent an alteration which takes place in the nervous tissue before the hæmorrhage and which itself may determine the hæmorrhage. The author thinks that lesions represented by the appearance of these cells may result in senile dementia in some people and in cerebral hæmorrhage in others. R. G. G.

[80] **The diagnostic value of sodium fluorescein in epidemic cerebrospinal meningitis.**—B. E. Bonar and L. S. Bailey. *Amer. Jour. Dis. Child.*, 1930, xl, 492.

The studies of Kafka, Schönfeld and Jervell have established the fact that sodium fluorescein given by mouth in doses of about 3 gm. per patient or ·05 gm. per kilogram of body weight will not permeate the meninges of the normal adult in two and a half hours. The workers studied sodium fluorescein as a diagnostic agent in twenty-one cases of cerebrospinal meningitis and in all but two fulminating cases the sodium fluorescein was found in the cerebrospinal fluid within two and a half hours after administration.

The test would appear to be of value in the early differential diagnosis of cerebrospinal meningitis from other conditions in which there are sometimes manifestations of meningeal irritation—pneumonia, erysipelas etc.—particularly should such cases occur during an epidemic of cerebrospinal meningitis. A. G.

[81] **Nitrite reaction as a diagnostic test in influenzal meningitis.**—Roy M. Greenthal. *Amer. Jour. Dis. Child.*, 1930, xl, 569.

The ability of *B. influenzæ* to reduce nitrates to nitrites may be used as a rapid diagnostic test in influenzal meningitis. A positive nitrite test, according to the method described, was obtained in thirteen consecutive cases of influenzal meningitis. The test can be completed within four to eight hours of lumbar puncture and was always negative in meningococcal, streptococcal and tuberculous meningitis. A. G.

[82] **Experimental combined system disease.**—E. F. Gildea, E. E. Kattwinkel, and W. B. Castle. *New England Jour. of Med.*, 1930, ccii, 523.

The authors' summary is largely as follows :

1. Eight dogs fed with a diet deficient in both the antineuritic and the ' pellagra-preventing ' elements of vitamin B, and six fed with the former only, developed loss of appetite, vomiting, and a spastic type of paralysis (posterior limbs), progressing rapidly to convulsions, coma, and death. Four given a diet lacking only the pellagra-preventing component developed no significant neurological symptoms, but died eventually in convulsions.

2. Post-mortem it was found that the 14 dogs of the first two groups had all suffered from a diffuse, irregular loss of myelin, chiefly in the white matter of the cord, and less marked in the cerebral cortex. In nine, slight lesions of the peripheral nerves were also noticed. The remaining four animals (third group) showed early changes in the cord.

3. The lesions of those in the first two groups undoubtedly bore a close resemblance to those of human subacute combined degeneration.

4. Gastric analyses disclosed no significant changes in the course of these observations and thus no evidence for a causal relationship between combined system disease and achlorhydria was established.

J. S. P.

[83] **The congenital morphological anomalies of the cranium and the vertebral columns in hereditary ataxia** (Le anomalie morfologiche congenite del cranio e della colonna vertebrale nelle atassie ereditarie).—U. DE GIACOMO. *Riv. di pat. nerv. e ment.*, 1929, xxxiv, 364.

BESIDES the phenomenon of bony fragility noted by the author in three individuals affected by hereditary ataxia, he has also constantly observed the presence of congenital morphological anomalies of the cranium and vertebral columns. The cranial anomalies were represented by a more or less notable narrowing of the posterior cerebral fossa, which corresponds to the hypoplasia of the cerebellum contained in it. The spinal anomalies were not confined to the cerebral end of the column, but were well-marked in the lumbar region and consisted of the presence of a supernumerary vertebra in one case, and sacralization of the fifth lumbar vertebra. The changes would seem to be due to an embryological bony dystrophy associated with the maldevelopment of the neighbouring nerve structures.

R. G. G.

[84] **Contribution to the study of vital coloration in the nervous system** (Contributo allo studio delle colorazioni vitali nel sistema nervoso).—C. BELLAVITIS. *Riv. di pat. nerv. e ment.*, 1929, xxxiv, 348.

THE author studied the vital coloration of the nervous system with injections of big doses of trypan blue in rabbits after having tried to irritate the neuroglia with nucleinate of soda or by injecting into the cerebral tissues paraffin as a foreign body. With the first method the results were negative; with the second, coloration occurred in the meninges, in the adventitious vessels, in the plexuses and the satellite cells, and the cells of the adipose granular layer. These reactions were especially abundant in the immediate neighbourhood of the wound. This is taken as a further proof of the mesodermal nature of the microglia.

R. G. G.

[85] **The chemistry of degenerating nervous tissue** (La chimie de la substance nerveuse au cours de sa dégradation).—Raoul M. May. *L'Encéphale*, 1930, xxv, 447.

The author reviews the older work of Koch, Mann, Mott and others on the gross chemistry of the brain and adds some experiments which he has himself performed by microchemical methods. Of special interest in his review of the literature are the observations of Delaville and Tcherniakofsky that in patients with anxiety states and in epileptics the chlorides in the blood rise with the crisis, the rise being especially in the intracorpuscular chlorides. Corresponding with this they found a great increase in intracerebral chlorides in a case of dementia paralytica and in a case of organic dementia. The author's own experiments do little more than test the applicability to this form of research of the micro-methods of Pregl. Unilateral lesions of the cerebrum of a guineapig and of the sciatic nerves of rabbits were examined after varying intervals, and a similar sequence of chemical change was found in both series of experiments. The total sulphur and nitrogen rose and the total phosphorus fell, the water remaining constant or rising slightly on the injured side. In spite of the fall in the total phosphorus, there was found to be a rise in the fraction of phosphorus soluble in water which included the phosphates and the simple organic compounds of phosphoric acid. The chief fall was in the lipoid and protein phosphorus compounds, and in a fraction soluble in alcohol but insoluble in ether or water. Translated into the more familiar terms of histology, these experiments indicate no more than a disintegration of myelin and phosphorus-containing nervous elements: and while they appear to demonstrate the utility of microchemical methods for this form of research, they do not lend any support to the author's claim that the chief advances in neuropathology in the future are likely to come from the gross chemical examination of diseased nervous tissues.

J. G. G.

SENSORIMOTOR NEUROLOGY.

[86] **Clinical and anatomo-pathological contribution to the study of tumours of the temporal lobe** (Contributo clinico ed anatomo-patologico allo studio dei tumori del lobo temporale).—R. Bozzi. *Riv. di pat. nerv. e ment.*, 1929, xxxiv, 429.

A long and comprehensive review of the subject. A personal case is fully discussed and the author concludes that the ophthalmoplegic factors of these cases depend on lesions of the oculomotor nuclei and the fibres derived therefrom. The alterations come into the category which other authors have described in cerebral tumours and used to explain certain symptoms, viz., 'action at a distance.' This may be partly due to general toxic action, but may

also have reference to some selective factor evolved in the life of the tumour.

The author concludes that the syndrome of temporal lobe tumour comprises the following :—

1. The general symptoms of cerebral tumour.
2. The local symptoms of sensory aphasia if the tumour is on the left.
3. Neighbouring symptoms and signs of disturbance of the pyramidal tract.
4. Symptoms of distant effects, lesions of the third nerve and cerebellar components.

The syndrome of some of these tumours is very vague and confined to a few psychical disturbances, but the author thinks that such are in the minority, and that localisation in respect of temporal neoplasms is becoming more defined.

R. G. G.

[87] **The tumours of the third ventricle** (Nueva contribucion sobre los tumores del tercer ventriculo).—J. F. Fulton and P. Bailey. *Archivos Argent. de Neurol.*, 1930, v, 3.

Symptoms referable to the region of the third ventricle are apt to be overlooked or disregarded when the sella turcica is reported normal. The differential diagnosis between tumours of the third and of the fourth ventricle is difficult since each may give the symptoms of the other.

Polyuria, adiposity, hypersomnia are among the common indications of a third ventricle neoplasm, to which the authors now add the occurrence of Quincke's œdema. Ventriculography is of great service in precise localisation. If the tumour is inoperable X-ray radiation sometimes effects remarkable clinical improvement.

S. A. K. W.

[88] **Forced grasping and frontal lobes** (Zwangsgreifen und Stirnhirn, sowie einige Bemerkungen über das occipito-frontale Bündel).—P. Schuster and J. Casper. *Zeits. f.d.g. Neurol. u. Psychiat.*, 1930, cxxix, 739.

This long article contains matter of considerable neurological interest and may briefly be summarised.

Forced grasping arises through removal of inhibition in the motor system. It shows itself clinically when the pyramidal path is for practical purposes more or less intact physiologically. In all probability the inhibitory path is of cortical origin and appears to come from the first or upper frontal gyrus and perhaps also from the cortex of the neighbouring gyrus fornicatus—lying mainly on the mesial aspect of the hemisphere.

According to the authors' researches the phenomenon develops in recognisable degree on *one* side as soon as the mechanism controlling grasping is inter-

fered with from *both* frontal lobes. In the majority of their cases the lesion was so situate as to involve both frontal lobe and corpus callosum and hence to effect the double action just mentioned.

It makes its appearance on the side opposite to the ' blocked ' hemisphere, but is sometimes accompanied by a much milder degree of the same disorder on the homolateral side. No clear evidence points to the predominance of one frontal lobe over the other in this connexion.

For the conduction of the inhibitory influence the occipito-frontal tract seems to be of significance, and impulses are transmitted to the opposite side via the corpus callosum.

S. A. K. W.

[89] **Paraplegia in flexion in consequence of a decerebration above the nucleus ruber.**—W. J. C. VERHAART. *Psychiat. en Neurol. Bladen*, 1930, xxxiii, 33.

THE case described is that of a woman of about 70 who in 1914 had a cerebral attack (right hemiplegia with some aphasia). A similar attack, with the same consequences, came 18 months later. Some three years before her admission to hospital the legs had become progressively weak and then powerless, and later the arms were affected. Symptoms of organic dementia also made their appearance.

Towards the end the clinical condition was as follows : the patient lay half on her left side, both legs sharply flexed at knee and hip, the right arm adducted and flexed at the elbow. Only the left arm was capable of any movement, the other limbs being paralysed. Tonus was everywhere increased : the knee-jerks could be obtained but were very slight ; the plantar reflexes were always doubtful. No ankle clonus could be elicited. On the other hand, the reflexes of ' defence ' were always obtainable in the legs.

The pathological condition proved to be one of severe cerebral arterio-sclerosis, with a general cortical and subcortical atrophy—mainly the result of numerous small necrotic foci and hæmorrhages. Lenticular nuclei and to a less extent the thalami were greatly reduced in volume. On the other hand, the anterior thalamic nuclei, nucleus ruber, corpus Luysii, and substantia nigra were for practical purposes normal, while structures at lower levels showed no primary injury.

The case may be considered one of combined pyramidal and extrapyramidal character. Decerebration down to the level of the pars magnocellularis of the nucleus ruber allows of paraplegia in flexion ; if the lesions extend further to include the whole of the nucleus, decerebrate rigidity of the usual type will develop. But the same paraplegia in flexion may be derived from lesions involving frontal and central gyri only, so that the basal ganglia presumably take no part in its onset. Regression to the flexed type is caused by

destruction of the frontal lobes and is accompanied by paralysis if the pyramidal systems are also involved. Lesions lower down effect no change in this condition till the level of the pars magnocellularis is reached, when the type changes to that of full decerebrate rigidity.

S. A. K. W.

[90] **Substitution in pyramidal function** (Über Ersatz der Pyramidenbahnfunktion).—J. VAN DER BRUGGEN. *Deuts. Zeits. f. Nervenheilk.*, 1930, cxiii, 250.

THIS interesting communication is based on a case of absolute crossed pyramidal degeneration on one side (infantile cerebral hemiplegia), in which none the less some functional power and movement existed on the affected side. There was definite evidence of hypertrophy of the sound pyramidal system. Several modes of functional substitution are sketched ; these include more use of the direct pyramidal tract, or of homolateral pyramidal fibres ; a further suggestion is abnormal division at the crossing. A number of references to similar cases are given.

S. A. K. W.

[91] **Does a syndrome of the subthalamic body of Luys exist ?** (Esiste una sindrome del corpo subtalamico di luys ?).—U. POPPI. *Riv. di pat. nerv. e ment.*, 1930, xxxv, 219.

THIRTEEN cases are reported from the literature of hemichorea, 11 of which showed lesions in the subthalamic body of Luys. In these the chorea was of great violence, a feature described as ' hemiballismus.' This particular variety of chorea is ascribed to the above lesion. In six there seemed to be a diffuse lesion of the extrapyramidal motor system and especially of the cerebello-rubro-thalamic system. The author thinks that lesions of the subthalamic region may cause different symptoms in experimental animals and in man ; and that vegetative functions belong to many of the nuclei, including that of Luys, in this region. He thinks that the violent chorea is probably due to interference with a neuronic system, possibly the cerebello-rubro-thalamic tract.

R. G. G.

[92] **The syndrome of the corpus Luysii** (Über das Syndrom des Corpus Luys an Hand eines anatomisch untersuchten Falles von Hemiballismus).—K. BALTHASAR. *Zeits. f.d.g. Neurol. u. Psychiat.*, 1930, cxxviii, 702.

THE case described is that of a woman of 65 who exhibited the syndrome of hemiballismus on a basis of cerebral arteriosclerosis. A softening was found

involving the right corpus Luysii but spreading beyond its anatomical limits. The writer argues for that structure being considered the essential anatomical factor in the production of the syndrome. Yet he admits that not every case of disease of the corpus is followed by hemiballismus, and contends that only strictly limited lesions will have that consequence. He does not accept the view that the clinical phenomena belong to the 'irritative' class, but considers them in the 'release' category. The normal function of the corpus is thought to be one of 'special movement-activities within the extrapyramidal mechanism'—whatever that may mean. S. A. K. W.

[93] **Clinical and genealogical observations in a case of the spastic pseudosclerosis of Jakob** (Klinische und genealogische Beobachtungen bei einem Fall von spastischer Pseudosklerose Jakobs).—F. MEGGENDORFER. *Zeits. f.d.g. Neurol. u. Psychiat.*, 1930, cxxviii, 337.

THE clinical features of the case, that of a woman of 46, include: gradual onset, after indefinite neurasthenia-like complaints, of pyramidal signs, coupled later with symptoms of striatal type: amnesia, severe dementia, and complete helplessness, mental and physical.

Its pathological basis is described in the paper by Stender (see below). S. A. K. W.

[94] **Further contributions on the subject of Jakob's spastic pseudosclerosis** (Weitere Beitrage zum Kapitel 'spastische Pseudosklerose Jekobs').—A. STENDER. *Zeits. f.d.g. Neurol. u. Psychiat.*, 1930, cxxviii, 528.

THE enamination in the case reported by Meggendorfer (above), and in two others, is summarised as follows: severe degenerative processes throughout the cerebral cortex and the basal ganglia, leading to sclerosis: marked fatty changes in cells, with remarkable proliferation of the protoplasmic glia. On the whole, the motor areas, frontal lobes, striatum and medial nuclei of thalamus are most involved; to a less extent, parietal and timporal cortex, globus pallidus and insula.

For anatomical reasons the condition should be separated from senile parenchymatous affections and from general paralysis. Of its etiology the author remarks that it may arise on a basis of heredo-degeneration, but cannot say whether this of itself suffices. S. A. K. W.

[95] **Observations on visceral pain.**—R. D. RUDOLF and A. G. SMITH. *Amer. Jour. Med. Sci.*, 1930, clxxx, 558.

THE authors deal only with the referred part of visceral pain and describe how in cases of angina pectoris, cholecystitis, duodenal ulcer and subacute appendi-

citis this was completely relieved by local intracutaneous injections of a 2 per cent. solution of novocain ; in many of their cases the true visceral part of the pain seemed also to be lessened at the same time. Thus superficially referred pain from a viscus is not a matter of central nervous projection, for it is abolished by local anæsthesia. They conclude that for the appreciation of referred pain from a viscus an intact reflex arc including the somatic portion is essential—the somatic neurones must be able to conduct normal afferent impulses from their periphery before referred visceral pain can be experienced in their distribution.

They noted too that deep discomfort was often lessened by surface anæsthesia, and suggest that the absence of parietal afferent impulses lessens the excitability of the visceral neurones—that afferent impulses from the source of visceral irritation are amplified to some extent by cutaneous impulses from the corresponding somatic segment.

A. G.

[96] **Determination of intracranial tension by Bailliart's ophthalmodynamometer** (Sur la détermination de la tension intracranienne par l'ophtalmodynamométrie de Bailliart).—Knud Winther. *Acta Psychiat. et Neurol.*, 1930, v, 403.

With Bailliart's ophthalmodynamometer, applied on the globe of the eye, it is possible so to augment intraocular pressure as to obtain pulsations of the central artery of the retina and by increasing it to cause them to cease. These diastolic and systolic pressures provide an index to intracranial pressure.

Full details are supplied of the instrument itself and of the necessary technique. By its means valuable corroboration of results obtained by spinal manometry can easily be procured. Many clinical data are recorded in the article.

J. V.

[97] **Optic nystagmus.**—Raymond Dodge, R. C. Travis and J. C. Fox. *Arch. of Neurol. and Psychiat.*, 1930, xxiv, 21.

By means of their instrument—the nystagmograph—the authors studied the characteristics of the slow phase of optic nystagmus. It was found that the slow phases are essentially eye-movements of pursuit, and may be termed adequate when the point of regard consistently falls on one of the moving objects of interest ; inadequate when the point of regard overshoots or lags behind the visual object.

With increasing speed of oscillation the rapid corrective eye-movements first increase and then decrease in both number and amplitude, occasionally reaching still fixation.

R. M. S.

[98] **Syndrome of post-choreic rigidity with dementia** (Le syndrome de rigidité post-choréïque avec démence).—H. CLAUDE, J. LHERMITTE, and P. MEIGNANT. *L'Encéphale*, 1930, XXV, 417 and 493.

THIS paper is divided into two parts, the first of which is the report of a case with pathological findings. At the age of 36, following a fall from a balloon in which, in spite of a parachute, he suffered some injury, the patient began to have constant choreiform movements. He became progressively insane and eventually demented so that he had to be certified at the age of 47. When examined soon after this he was quite unable to walk, to speak, or to feed himself. His limbs and trunk were rigid, but the muscles were not hard and the contraction was variable. When he stood on his feet choreiform movements were much more evident. There was no propulsion or retropulsion but when pushed in any direction the patient fell to the ground unless supported. Although the tendon reflexes were brisk and perhaps exaggerated the plantars were definitely flexor in character. None of the Magnus and de Kleijn reflexes could be elicited.

In the month which elapsed between this examination and the death of the patient there was still more disappearance of the choreiform movements and a greater tendency to fixed symmetry of the position of the limbs.

At autopsy there was general shrinkage of the brain, affecting the cortex diffusely so that all the sulci were widened. The basal ganglia also were noticably shrunken, but no softenings were seen anywhere. Histologically a considerable loss and sclerosis of nerve cells was seen throughout the cortex. Still more evident was the disappearance of the small nerve-cells of the corpus striatum. The larger nerve-cells in the putamen-caudate and in the globus pallidus were also greatly diminished in number, but not to the same extent. There were moreover in the corpus striatum and globus pallidus inflammatory lesions of the nature of perivascular infiltration and inflammatory nodules. Apart from some degeneration of the cells of the substantia nigra the brain-stem was normal and the changes in the optic thalamus were slight.

In the second half of their paper the authors discuss the relationship of chorea and rigidity to lesions of the basal ganglia. Two other cases of rigidity following on chorea have been described, one by Bielschowsky, the other by Jakob. In Bielschowsky's case the disease began at the age of six. Rigidity supervened on choreiform movements after the age of nine and death occurred at the age of 14. The most striking histological feature in this case was the loss of the small cells of the putamen-caudate, but there was also considerable atrophy of the large cells of the corpus striatum and globus pallidus. Cellular lesions were found in the corpus Luysii, the rèd nucleus and the substantia nigra, and degeneration of the cortical nerve-cells was also manifest.

Jakob's case was that of a man who came into hospital at the age of 47 with chorea and died five years later with progressive rigidity during the last year of his life. In this case also there was considerable loss of cells throughout

the globus pallidus and corpus striatum but especially in the caudate nucleus. The substantia nigra was also affected. The authors in a review of the literature of progressive chorea discuss the various views which have been held as to the causation of chorea by lesions of one or other part of the basal ganglia. They are inclined to accept the view that rigidity is brought on by a lesion of the large cells of the globus pallidus and corpus striatum and that choreiform movements may be related to loss of the small nerve-cells of the corpus striatum. In the cases cited the spread of the disease to the globus pallidus seems to have produced muscular rigidity and at the same time to have abolished the choreiform movements. They hold that Foerster's observations disprove the relationship of cortical lesions to chorea. The mechanism by which these modifications of muscular control are brought about is not yet at all fully understood, although various theories have been put forward to explain them. They accept, however, as broadly true, the view that the corpus striatum and globus pallidus are motor organs subserving automatic movements; the corpus striatum being a complex organ controls the activity of the more simply constituted globus pallidus, and both control the lower centres. But the results of lesions of these centres may depend not only on their topography but also on their quality. Some may be due to excitation of nerve-cells, others to their destruction, and the same or similar symptoms may arise from excitation of one group of cells or from destruction of the cells which normally control them.

Although in this part of their paper the authors have done little more than discuss theories, the association of their case with others in which a similar clinical picture was associated with a lesion of similar nature and topography makes it unusually interesting and important.

J. G. G.

[99] **A case of 'hormetonia' [Davidenkoff] resulting from an intraventricular hæmorrhage** (Un cas d'hormétonie de Davidenkoff consécutif à une hémorragie cérébrale ventriculaire).—Theodore Dosuzkov. *L'Encéphale*, 1930, xxv, 302.

The case reported is that of a woman of 63 who two years before her final attack had suffered from a stroke with right hemiplegia and some aphasia. The final attack, which was fatal in two and a half hours, was unusual in that within a few minutes of the stroke the limbs on both sides, but particularly on the right, were in a state of considerable hypertonus. A 'fan sign' appeared on both sides and a grasp reflex in the right hand. After half an hour hypertonus was replaced by hypotonus; this again was later replaced by hypertonus, and so on until her death. Post mortem the old lesion was present as a focus of softening near the vertex of the parietal lobe, which spared the internal capsule. Old hæmorrhages were also present in the cerebellar hemispheres, affecting among other structures both dentate nuclei. The new

hæmorrhage had burst through the head of the right caudate nucleus and optic thalamus into the third ventricle, destroying also the inner surface of the left optic thalamus, and spreading into the iter and third ventricle.

The author discusses the syndrome of hormetonia described by Davidenkoff in 1919. This consists of alternating hyper- and hypotonus in the paralysed limbs, coming on immediately after a stroke. It is associated with defence reflexes and postural neck reflexes and a loss or at least great diminution of consciousness. It appears to be caused by various severe lesions of the hemispheres or brainstem, and is considered by Davidenkoff to be the human analogy of decerebrate rigidity in animals. J. G. G.

[100] **Pathological anatomy and physiology of Sydenham's chorea** (Anatomie et physiologie pathologiques de la chorée de Sydenham).—J. Lhermitte and Ph. Pagniez. *L'Encephale*, 1930, xxv, 24.

The case described here is that of a girl of 19 whose movements had become progressively choreiform for six weeks before death. It is noteworthy in view of the pathological findings that the patient had been given intravenous injections of 2 and 3 grammes of sodium salicylate, and that during the 48 hours which preceded death the temperature had risen gradually to 40° C. The most noteworthy alteration in the nervous system was a diffuse chromatolytic lesion of the nerve-cells, which affected in greater or less degree all the cells examined from the medulla to the cortex. Slight neuroglial changes and some perivascular œdema were also noted, but no inflammatory changes were found. The case thus gives little help in the elucidation of the vexed problem of the pathogenesis of chorea minor. There appear to be two groups of cases, one in which inflammatory lesions have been found in the basal ganglia and cortex, the other in which the lesions are of a purely toxic or degenerative character. In the present case they are so diffuse as to suggest that they were rather due to the manner of death than that they bore any relationship to the preceding symptoms. The authors criticise at some length the theories of Kinnier Wilson on the cortical origin of chorea, and consider that lesions of the basal ganglia or of the cerebellum may play a large part in the production of choreiform movements. J. G. G.

PROGNOSIS AND TREATMENT.

[101] **Further contributions to the chemical induction of hyperpyrexia in the treatment of general paralysis and other diseases of the neuraxis** (Ulteriore contributo alla piretoterapia chimica nella paralisi progressiva ed in altre malattie del nevrasse).—P. Armenise. *Riv. di pat. nerv. e ment.*, 1930, xxxv, 326.

The author has treated 12 cases of general paralysis, 3 of cerebral syphilis and 7 of dementia præcox by supraperiosteal injection of oleated sublimed

sulphur. He obtained complete remissions in six of the cases of general paralysis and in one of cerebral syphilis, and slight improvement in five other patients. He noted a certain therapeutic action, of no great extent, in some cases of dementia præcox. R. G. G.

[102] **Clinical observations on malaria by inoculation from the point of view of ordinary malaria and its parasitology** (Osservazioni cliniche, malariologiche e parassitologiche sulla malaria da inoculazione).—A. Mari. *Riv. di pat. nerv. e ment.*, 1929, xxxiv, 672.

The clinical symptomatology of malaria by inoculation is the same as that of malaria acquired by the ordinary channels. The irregularity of the fever in the former type is not due to the passage of the parasite from man to man, but to the peculiarities of the strain used. There is no convincing evidence of the existence of a spontaneous and acquired immunity for malaria. Malaria by inoculation, whether benign, tertiary, or quaternary, does not show relapses if enough quinine is used.

The bodily and hæmatological manifestations are the same however the malaria is acquired. There are no variations in the morphology or life-cycle of the parasite brought about by numerous passages from man to man. Gametocytes may be produced in both, and are disseminated in the same way. R. G. G.

Psychopathology.

PSYCHOLOGY.

[103] **Emotions in terms of the galvanometric technique.**—Christian A. Ruckmick. *Brit. Jour. Psychol.*, 1930, xxi, 149.

It may be concluded that the galvanic reflex, when properly safeguarded against both uncontrolled emotions within the body that are foreign to the emotional situation and the fortuitous factors in the electrodes, affords us a promising approach to a quantitative and graphic analysis of the emotional life. We can say with some degree of certainty that we have a means of graphically recording the temporal course of an emotional process with its intensity closely corresponding to the amount of variation in bodily resistance. We may say further that the most primitive and instinctive emotions, i.e. those having large bodily components in terms of kinæsthesis or organic sensations, more definitely show graphic disturbances: emotions that are full of ideational material, on the

other hand, give much more reduced deflections. In any case the disturbances accompanying emotional experiences can ordinarily be distinguished from bodily responses as a whole through the appearance of the initial latent period followed by a rapid incidence of the curve of deflection. C. S. R.

[104] **Is there a 'time' sense?**—A. O. JONES. *Psyche*, 1930, xi, 63.

THE writer possesses the faculty of being able to wake at any predetermined hour and also of being able to state the time—with considerable accuracy—on being asked. Concerning the latter he has observed some singular points. As far as he knows it is in no sense guess work. In fact, if, before answering, he thinks what the time is likely to be, he is inaccurate by wide amounts. Again, if it has so happened that a short time before being asked he has looked at his watch and noted the time, and then, conscious of that fact, is asked, and tries to estimate the time, he finds himself very inaccurate in his reply. But the unpremeditated answer is accurate.

This faculty appears to the writer to differ somewhat from that of being able to wake at a predetermined hour. The latter was upset by summer time, the former was not. He finds it difficult to advance any satisfactory explanation of it other than on the lines that man may possess a definite time sense, which is suggested also by certain post-hypnotic experiments. C. S. R

[105] **The psychology of sea-sickness.**—CLAUDE A. CLAREMONT. *Psyche*, 1930, xi, 86.

WE are accustomed to receive certain sensations from the eyes in conjunction with others from the soles of the feet, joints, back, or other points of support. Normally the two sets agree with one another, or rather their occurrence in a given conjunction we are accustomed to regard as normal. This normality is disturbed on board ship. Our eyes here tell us that we are stationary, since we are moving with the room. But our sensations of support will have it that we are moving. We feel changes of pressure as the ship goes up and down. Under all known conditions, sensations of that kind would be accompanied by movements of the room about us (relative to the eye). Here there are no such movements. Some sense or other, it seems, must be misleading us. The assumption that sea-sickness is due to the unaccustomed conflict between sensations normally combined in other ways will be found, so the writer thinks, to fit all facts. In swinging, the sensations of movement are in accord with the movements seen and anticipated by the eye. No sickness results. The situation is different on a ship. The horizon is too far away to serve as an indicator to the eye of relatively trifling movements with regard to it.

The view here advanced was known many years ago to Trousseau and others as 'cerebral surprise.' C. S. R.

NEUROSES AND PSYCHONEUROSES.

[106] **Masturbation: its role in the neuroses.**—ADOLPH STERN. *Amer. Jour. Psychiat.*, 1930, ix, 1081.

PRIMARILY a longtitudinal section of the sexual life is sketched out on Freudian lines. The pathological significance of masturbation is manifest in the third period of sexual development, viz., during and following the period of puberty. The harmful effects may be summed up under three headings. Firstly, in the physical field harm may result from excess and from the lack of gratification accompanying the act. The mechanism of the symptoms noted is difficult of explanation. According to Ferenczi, masturbation with fantasy as the chief, if not the only, stimulant, is an insufficient discharging mechanism for the excitations aroused. It still is difficult to understand the incidence of morbidity and some individual predisposition must be predicated. Secondly, because of the excessive fantasy formation, there is the danger that an individual so occupied will exert no effort to bring about changes in the external world, demanded by, and commensurate with, reality. Fantasy will form the main source of gratification and its prolongation into adult life adds to the difficulties under which the individual labours to renounce this form of gratification. Thirdly, masturbation, if it persists during and after adolescence, will tend to bring about a fixation of sexual aims and the persistence of psychosexual infantilism. With this condition established, the handicapped individual has the predisposition to neurosis well-established. Lastly, as pathological results, certain character traits will likely be noted. The chronic masturbator carries with him a feeling of guilt and fear of discovery. He becomes asocial, overserious, too conscientious, and the feeling that he is dishonest impels him to exhibit a fanaticism for honesty, virtue, and uprightness. C. S. R.

[107] **The nervous temperament.**—MAY SMITH. *Brit. Jour. Med. Psychol.*, 1930, x, 99.

FOLLOWING upon a survey of the development of industrial psychology showing the circumstances leading up to the study of the nervous temperament, the author deals with the historical background of the problem and the motives which have led to its continued existence as a problem. After recounting contemporary work done from the physiological aspect, Miss Smith's own investigations are recorded. She concludes that from the point of view of health and successful work, the diagnosis and treatment of the nervous person are important. She shows that nervous people on the average tend to have more sick leave, to be less efficient, and to suffer more from conditions that are neutral to others and, in some occupations, to develop an occupational cramp. Just as some people are physically unfitted for certain occupations and are

therefore kept out, and some are too intelligent or too unintelligent for some work, so there are people temperamentally unsuited for particular conditions, and they should be diverted into occupations suitable for them. C. S. R.

PSYCHOSES.

[108] **The examination of cases of dementia præcox during the three-year period, 1926–1928, by histoneuropathological and encephalographic methods and by examination of the cerebrospinal fluid** (Ricerche istoneuropatologiche, encefalografiche e del liquor in dementi precoci: Triennio 1926–1928).—V. M. Buscaino. *Riv. di pat. nerv. e ment.*, 1929, xxxiv, 181.

The author thinks that pathological changes of structure occur in the nervous system from the first days of the illness and that this can be recognized during life by encephalography and examination of the fluid. These changes consist of lesions in the meninges which are inconspicuous and variable, degenerative changes in the vessels, in the nerve-cells, in the glia and in the nerve fibres. The most characteristic changes are the areas of disintegration like bunches of grapes on which the author has done so much work. The lesions are focal and widely disseminated, chiefly in the cortex but also in all parts of the brain. The cortical lesions may occur anywhere but chiefly in the frontal lobe and in the third cortical layer. Regenerative processes also occur.

The nature of the lesions and their irregular distribution account for the irregularity of symptoms. Their predilection for the third stratum accounts for the intellectual-emotional dissociation, and the extracortical lesions for the catatonia.

The author concludes that though still unknown the cause of dementia præcox is a pathological problem and not constitutional.

R. G. G.

[109] **Some observations on the etiology of dementia præcox.**—W. S. Jagoe Shaw. *Jour. of Ment. Sci.*, 1930, lxxvi, 505.

After briefly reviewing the main current views of the causation of dementia præcox, the writer states that as a result of his experience of its peculiar prevalence among the Parsis of Bombay, he is convinced that in-breeding is an important factor. Since the Parsis left Persia in the eighth century, the intermarriage of cousins has been not only common but usual. Among the last 150 cases of mental disorder among Parsis the author has met with, no less than 76 were cases of dementia præcox. This singular prevalence is specially noticeable as the disease is comparatively rare among Hindus, who constitute by far the largest section of the population, and rarer still among Mohammedans.

The criticism that this prevalence of schizophrenia among Parsis was probably due to western education is refuted. It is conjectured that the continuous marriage of cousins in some way lessens the power of resistance to the stresses of life, producing the 'lack of vitality' (to use Mott's phraseology), and the disorders of metabolism, that are the exciting factors in the break-down.

C. S. R.

[110] **Mescalin catatonia and the experimental production of the manifestations of dementia præcox** (Über Meskalin-Katatonie und die experimentelle Erzeugung von Dementia præcox-Erscheinungen).—H. DE JONG. *Konin. Akad. van Wetensch. te Amsterdam*, 1930, xxxiii, 1076.

THE author has experimented on animals (apes, cats, mice) with mescalin and been able to produce phenomena of a catatonic kind in definite fashion. He couples with this work the data derived from mescalin intoxication in man, viz., a condition resembling toxic psychosis with hallucinations, together with such sympathetic signs as vomiting, salivation, and mydriasis. On this basis he argues for the possibility of the production of a symptomatological triad closely resembling that of dementia præcox; he believes the approximation to the acute stages of schizophrenia is unmistakable. On the other hand, it is not to be expected that mescalin should be able to reproduce the syndrome of that psychosis in all its details.

S. A. K. W.

[111] **Post-prohibition alcoholic psychoses in New York State.**—WILLIAM C. GARVIN. *Amer. Jour. Psychiat.*, 1930, ix, 739.

A MARKED decline in first admissions of cases of alcoholic psychosis to the New York State Hospitals has taken place during the past two decades. The lowest admission rate occurred in the fiscal year ending June 1920, viz., 1·9 per cent. Since 1920 there has been a gradual rise in admissions, the peak of the curve being reached in 1927, viz., 7·0 per cent. In 1928 the rate dropped to 5.9 per cent. The reduction in the admission rate has been relatively greater among women than among men. First admissions with alcoholic psychoses came principally from urban districts. These psychoses were most prevalent in the Irish and Slavonic races, and were comparatively rare with the Jews. They, as a rule, occur principally in middle age, following years of intemperate use of alcohol. There has been a general reduction in the intemperate use of alcohol among all first admissions to the New York State Hospitals from 1911 to 1927. Acute hallucinosis is the outstanding type of alcoholic psychosis. There has been observed a change in the symptomatic picture as compared with the pre-war type, in that the patients appear more toxic and prostrated. There is often confusion, disorientation, combined hallucinations and delirious

admixtures during the acute phase. Recovery in the more severe cases is slower than in pre-war days. Korsakow's psychosis has decreased in frequency since the war and the mental symptoms and polyneuritis are much less severe. Permanent mental and physical residua are less common and recoveries more frequent than in the pre-war period.

C. S. R.

PSYCHOPATHOLOGY.

[112] **The pathological anatomy of chronic alcoholism** (Zur pathologischen Anatomie des chronischen Alkoholismus).—T. Ohkuma. *Zeits. f.d.g. Neurol. u. Psychiat.*, 1930, cxxvi, 94.

An elaborate histophathological study is based on the examination of material from four cases, with the phenomena of delirium tremens and of Korsakow's psychosis respectively in two.

The pathological foundation is one of diffuse, progressive, chronic parenchymatous nervous degeneration affecting a great part of the neuraxis. On this appears evidence of acute or subacute changes in the form of fresh capillary extravasations and glial reactions. In addition, however, must be noted the occurrence of local parenchymatous change, independently of the other, but of the same character, and usually, though not always, associated with vascular alterations. Vessel proliferation is seen with some frequency e.g., in corpora mamillaria, periventricular grey matter, neighbourhood of the aqueduct of Sylvius. It may be taken to represent efforts at tissue-repair. On the other hand, degeneration of vascular endothelium is often particularly prominent.

All the evidence goes to suggest that the noxa penetrates the nervous tissues from the cerebrospinal fluid, yet this in its turn does not clearly explain the involvement of deeper-lying structures; on the other hand, the position and nature of the processes in the vessels do not favour the view of entry by the blood-stream. The author with some diffidence takes refuge in the conception of a constitutionally influenced state of lowered resistance for certain parts of the neuraxis to alcoholigenic toxins, rather than to alcohol itself.

S. A. K. W.

[113] **The pathological anatomy of brain parenchyma at the involution period** (Pathologische Anatomie des Hirnparenchyms in der Involutionsperiode).—K. H. Bouman. *Psychiat. en Neurol. Bladen*, 1929, xxxiii, 423.

Four chief types of cerebral change can be separated in connexion with the mental symptoms of the period of involution.

1. In simple cases, an extremely slow cell atrophy develops, accompanied by fatty degeneration and mild sclerosis. Products of degeneration accumulate in the glia and along the course of the vessels.

2. Dementia of the senile variety is characterised by similar histopathological change, except that all the processes are more severe and more acute. Regressive modifications of the glia are more in evidence and cell atrophy becomes pronounced.

3. Alzheimer's disease is associated more especially with senile plaques and fibrillar degeneration, as well as with convolutional atrophy.

4. Pick's cortical atrophy is apt to affect principally frontal and temporal fields and the region of the insula.

S. A. K. W.

[114] **On the value of a rapid histological method for demonstrating senile plaques** (Sulla scelta di un metodo istologico rapido per la dimostrazione delle placche senili).—C. UGURGIERI. *Riv. di pat. nerv. e ment.*, 1930, xxxv, 265.

A SILVER nitrate method is described for the rapid demonstration of senile alterations which has the advantage over other methods of greater simplicity of technique, greater constancy of results, and higher selectivity for the senile plaques.

R. G. G.

[115] **The behaviour of the bilirubin in the blood in amentia and other mental illness** (Comportamento della bilirubinemia nell' amenza. ed in altre malattie mentali).—C. UGURGIERI. *Riv. di pat. nerv. e ment.*, 1929, xxxiv, 882.

THE authors have made in 72 cases of various forms of mental illness 127 qualitative and quantitative determinations of the bilirubin in the blood according to the method of Van den Bergh and have at the same time examined the resistance of the erythrocytes, the blood platelets and in the urine the urobilinogen and urobilin. The principal examination was in amentia, where the bilirubin was found to be increased in 100 per cent. of cases. The other conditions were examined as controls. It is notable that in amentia the increase of bilirubin corresponds with the increase of the activity of the somatic and confusional symptoms. Early cases of dementia præcox correspond in this respect with cases of amentia. It is suggested that a hæmolytic factor is the cause of the increase of bilirubin and is due to a lesion in hepatic cells.

While the state of the bilirubin cannot yet be considered as a specific diagnostic factor it is of important prognostic significance. R. G. G.

[116] **The genito-cutaneous reaction in the insane** (La geno-cutireazione negli alienati) —G. DE NIGRIS. *Riv. di pat. nerv. e ment.*, 1930, xxxv, 22.

THE genito-cutaneous reaction of Ceni, which is an allergic cutaneous reaction provoked by the active principles of the hyperactive genital glands of the lower vertebrates and which indicates the activity of the sexual glands of the individual, was tried in over 1000 insane patients and demonstrated frequent hypoactivity and weakness independently of age and of physical state, but in accordance with the mental condition. R. G. G.

[117] **Current conceptions of psychopathic personality.**—G. E. PARTRIDGE. *Amer. Jour. Psychiat.*, 1930, x, 53.

AFTER discussing the subject historically and from many angles, it is concluded that the views of psychopathic personality show wide disparity. In the widest sense the term is used to include all personality deviations of whatever kind. Closely related to the idea of the constitutional inferior, there has emerged a conception of forms of deviation which have as their essential trait deep and persistent sets in some direction, which produces persistent antisocial or socially futile behaviour. Persons of this type seem to have such a strong family resemblance one to another that it is tempting to think of them as displaying a deficiency in regard to some unit character. Their reactions show both identity of features and also widely variant traits and manifestations. Various individuals in this class, which are best called 'sociopathic,' resemble or approximate to other types of personality and the psychotic groups as well. The closest resemblance is probably to the paranoidal syndromes. They display also qualities which are closely related to the obsessional states, since they are always dominated by some fascination which takes an obsessional or compulsive form. It is not allowable, however, to regard these conditions as merely undeveloped psychoses, or undifferentiated psychoses of mild form, or to merge them forthwith into some expanded psychotic group. Their claim to self-identity is the consistent appearance of patterns which do not occur in other conditions. On the other hand to regard the psychopathic as differing in kind from the other mental deviations is equally misleading. What we should say of them is that the sociopathic developments constitute a type of maladjustment which does not usually show developments and episodes like those which appear as adjustment phases in the other conditions. The sociopaths adjust in a way that is characteristic of their class. None the less they are to be regarded as victims of mental disorder or disease. It is especially misleading to think of these conditions as different on the ground that they are only disorders of the personality, and that what they show is merely something

which is 'normal' for the individual.. Every one of the functional mental disorders may be regarded, from one point of view, as personality disorder—when considered longitudinally—and to regard the sociopathic types of reactions as uniquely personality disorders is not warranted.

C. S. R.

[118] **Axillary 'menstruation' in a male.**—ERNEST E. HADLEY. *Amer Jour. Psychiat.*, 1930, ix, 1101.

A HIGHLY interesting history is here given of a twenty-four year old male in whom a bloody seepage from the left axilla occurred during four to five day periods for at least seven regular monthly cycles. That such a physiological response may result from psychological sources is well known. This symptom was not the one which caused the patient to consult a psychiatrist: it was to him but additional confirmation of the belief that he was 'queer' and hopelessly insane. The sero-sanguineous material was slightly paler than the uterine secretions. After washing the armpit, unbroken skin was revealed and there was no ecchymosis. Gradually, droplets of the bloody or hæmoglobin-stained secretion reappeared. Treatment on psychoanalytic lines brought about a disappearance of this phenomenon as well as a recovery from his mental symptoms. This axillary 'menstruation' was regarded as a symbolic defence against sexual assault and innocence.

C. S. R.

[119] **Perseveration in the insane epileptic.**—JOHN H. EWEN. *Jour. of Ment. Sci.*, 1930, lxxvi, 537.

CONSIDERATION of experimental results seems to show that perseveration is a feature of the peculiar mental state of insane epileptics between attacks. The predominating factor in the associated psychosis naturally has a bearing upon the results, and in this connection it was of interest to contrast the findings obtained from similar tests applied to cases of mania and melancholia. The results in the motor and ideational tests were better in cases of mania than in melancholia according to Wynn Jones's findings, while the male epileptic results were better than the female epileptic results in the colour test, the motor test and the ideational tests. While differentiation on these lines is obviously inadequate, it is a point worthy of notice that all the male epileptics tested were prone to maniacal outbursts, while the female epileptics were more inclined to the melancholic syndrome. Taking the results as a whole, it would appear that mental inertia as measured by perseveration is one of the peculiarities of the epileptic constitution.

C. S. R.

PROGNOSIS AND TREATMENT.

[120] **Spinal drainage in alcoholic deliria and other acute alcoholic psychoses.**—HARRY GOLDSMITH. *Amer. Jour. Psychiat.*, 1930, x, 255.

SPINAL drainage is an effective means of treatment in acute alcoholic delirium states, attended by very little sequelæ : the length of stay in hospital of patients suffering with these conditions has been materially reduced in 78 per cent. of the cases to an average stay of only 30 days. Treatment is most effective when instituted within 24 hours following admission. No after treatment is necessary in the great majority of cases. They convalesce rapidly under routine hospital care.

C. S. R.

Reviews and Notices of Books.

Der bedingte Reflex. By Dr. N. E. ISCHLONDSKY, Paris. With 39 illustrations. Berlin and Vienna: Urban and Schwarzenberg. 1930. Pp. 326. Price R.M. 20.

ENGLISH translations have familiarised the neurologist and psychologist in recent years with Pavlov's monumental work on the conditioned reflex, and incidentally have rendered understanding of its abundant and technical literature less arduous. Pavlov himself has repeatedly if briefly indicated the possible applications of the doctrine to biological, medical, and psychological questions: and this is the main thesis of the volume under review. 'Experimental reflexology', as it is somewhat cumbrously termed, will also prove of service in paedogogy, according to Dr. Ischlondsky, whose general conclusions may briefly be sketched. The 'instincts' of the psychologist are nothing else than unconditioned reflexes: most, no doubt, are more complex than those capable of investigation in a physiological laboratory, but the difference is quantitative only, not qualitative. They may be described as complexes of common reflex reactions, serving a useful purpose for the animal concerned. No scientific classification of human instincts (innate reflexes) on reflex lines has as yet been carried out, for not enough is known of their origins: yet their genealogy must some day be elucidated, since knowledge of it is indispensable to an appreciation of the activities of the neuropsyche in man. For all living creatures the basic, fundamental reflex, from which all others are derived, is the reflex (or instinct) of life, which is independent of the organism's position in the animal scale, and holds true for man equally with the one-celled infusorian. The goal or aim of life is nothing other than the fulfilment of the demands of the life-reflex. Self-defence, nourishment, sexual, and orientation reflexes are dealt with, and others of no less significance. There is a great deal of physiological matter in the book of deep interest even if the author's deductions therefrom may sometimes appear a little strained. It should prove informative to all students of the conditioned reflex.

Physiologische Grundlagen der Tiefenpsychologie. By Dr. N. E. ISCHLONDSKY, Paris. With 15 illustrations. Berlin and Vienna: Urban and Schwarzenberg. 1930. Pp. 356. Price R.M. 22.

IN this second part of Dr. Ischlondsky's general work entitled 'Neuropsyche und Hirnrinde' a bold attempt is made to seek a physiological interpretation

for the phenomena of so-called deep psychology and in particular for the data of psychoanalysis. The mechanics, the dynamics, of human neuropsychical function are sought objectively, and however vague the idea of tracing an objective basis for manifestations in the realm of thought may at first seem, it is at least far removed from any correlation of mere morphology with psychology. With the statics, so to speak, of the problem the author is little concerned. His researches are erected on the fundamental conception that the conditioned reflex underlies neuropsychical function. Excitation and inhibition processes are opposed to each other, and according as one or the other prevails in given circumstances will its expression be apparent in the field of consciousness. When equilibrium obtains between the two, under conditions here fully examined, states of sleep, hypnosis, catalepsy and others of an analogous kind develop, according as the balance is normal or abnormal. An effort is made, ingeniously suggestive, to explain the function of speech on conditioned reflex lines.

The major portion of the book, however, is devoted to a consideration of psychoanalytic conceptions and facts from a similar standpoint, and certain 'classical' cases in the history of Freudian doctrine (e.g. those of Elisabeth von R. and Cäcilie M.) are re-examined with this end in view. Instead of tracing a chain of psychical links in the causation of the symptoms the author finds for each of these a dynamic, physiological explanation in the play of conditioned reflexes. In general terms, for example, a neurotic symptom is not immediately expressive of a 'suppressed affect' but of an 'inhibition lesion.' To follow him through all the working out of his thesis is for reasons of space impracticable, but the reader cannot fail to appreciate the author's skill in its application, nor can he quarrel with his main argument, that if physiological research should not meddle with events of the psychical series all the phenomena of nature, however complicated and enigmatic, are capable of being conceived from the standpoint of time and space, and their mechanisms of origination and development similarly. In other words, an objective basis for psychical events can be made the subject of exact, scientific, methodological investigation.

Grundlagen und Entwicklungsgeschichte der kindlichen Neurose. By Dr. ERICH BENJAMIN, Professor of Children's Diseases in Munich. Leipzig: Georg Thieme. 1930. Pp. 144. Price R.M. 9.

PROBLEMS of neurosis in children are being assailed at home and abroad by a host of investigators, and this latest contribution on the 'nervous child' has features of its own which will interest both physicians and teachers. The attention of the writer is focussed mainly on the 'Trotzperiode'—the period between two and four years when the child is prone to exhibit traits of defence and defiance against the demands and claims of others older than himself. Towards the end of this time of stress comes what the writer calls the child's

first puberty,'—about the age of three and a half—when a series of symptoms (requiring some 15 lines for catalogue) of a neurotic kind may be expected, most of which are coloured by an erotic tone. The argument here advanced is to the effect that should the symptoms of these times persist beyond their appropriate period they ipso facto constitute evidence of youthful neurosis. A distinction is drawn between their relative incidence on the two sexes, and statistics are given which go to prove that they linger longer in the boy than the girl. It is held that the clue to the neuroses of later years is to be sought in the reactions of these pre-puberty epochs.

Aphasia in Children. By A. W. G. Ewing, M.A., Ph.D. With an introduction by E. D. Adrian, M.D., F.R.S. Humphrey Milford: Oxford University Press. 1930. Pp. 152. Price 10s. 6d. net.

To the neurologist the title of this book is rather misleading, for the author does not deal with aphasia as a whole but confines his attention almost entirely to a series of cases in which a condition of partial deafness was discovered. In some respects they would be classified by the neurologist as instances of congenital auditory imperception: and it is of considerable interest and no little importance to note that Dr. Ewing was able to demonstrate in six out of ten of his cases a condition of normal hearing for a certain range of low tones coupled with marked deafness to high tones. For these he has devised the expression 'high-frequency deaf.' That the auditory defect was at least in some degree responsible for the clinical condition seems clear.

Of the four other cases studied Dr. Ewing remarks that their clinical state differed radically from that "due to injuries of the brain, studied by other workers and described as 'aphasia,'" and he prefers the term 'linguistic retardation.' Whether this distinction is justified by his description of their symptoms is open to question.

The Psychology of Insanity. By Bernard Hart, M.D., F.R.C.P. Fourth edition. Cambridge University Press. 1930. Pp. 176. Price 3s. net.

A book originally written in 1912 and reprinted at intervals ever since, with additions and modifications to modernise it, needs no commendation from us. Dr. Bernard Hart's little volume must surely be known wherever the mind diseased is studied, and that means, universally. The combination of clear thinking with graphic writing herein manifested proves irresistible, as the wide circulation of the book shows. In view of the continuing demand for it, we should like Dr. Hart to consider whether in his explanation of hysterical phenomena (p. 83) incongruity as between conflicting mental processes—a *general* condition—really is adequate to explain the *specificity* of numerous hysterical symptoms. And if (p. 44) retrograde amnesia is characteristic of

hysteria, why not insert that it is equally frequent in cerebral concussion? Emotional phenomena in connexion with incurable cancer, and spes phthisica (p. 105), are assigned by Dr. Hart a psychogenic origin; perhaps some day he will allow that they are equally capable of explanation from a purely physiological standpoint.

Psychopathology. By BERNARD HART, M.D., F.R.C.P. Second edition. Cambridge University Press. 1930. Pp. 178. Price 8s. 6d.

ON its first appearance this volume received favourable review in this JOURNAL (Vol. VIII, p. 86), and in its new form is sure of further appreciation. The second edition includes an added chapter on 'The Conception of Dissociation,' amplifying the author's views in regard to the respective contributions to psychopathology of Janet and of Freud. Briefly, the book constitutes a detailed and well-considered review of the development of psychopathology since the beginning of the century and of its present place among the subdivisions of medicine; it also contains discussions on 'The Psychology of Rumour' and on 'The Methods of Psychotherapy.'

Grundzüge der Neurochirurgie. By Prof. Dr. WALTER LEHMANN, Frankfürt. (Vol. VIII, Medizinische Praxis, edited by Prof. Dr. L. R. GRÖTE, Prof. Dr. A. FROMME, and Prof. Dr. K. WARNEKROS.) With 28 illustrations. Dresden and Leipzig: Theodor Steinkopff. 1930. Pp. 197. Price R.M. 13.50.

IN convenient form, well printed and illustrated, Prof. Lehmann offers the practitioner and neurologist a useful and up-to-date description of the procedures of neurosurgery, which covers a remarkably wide field. The neurological surgeon has been steadily pushing his researches into regions formerly considered inaccessible, and while his major operations have increased in range of possibility, those of a minor class are no less important from the standpoint of therapeutics. In this handy volume will be found accounts of all present-day methods, including work on the sympathetic system, epilepsy, neuralgia, spastic conditions, etc. A book of this class may be cordially recommended.

La Neurosyphilis. By A. RADOVICI, Lecturer in Neurology, Faculty of Medicine, Bucarest. Paris: Masson et Cie. 1930. Pp. 365. Price 40 fr.

DR. RADOVICI has written a meritorious synthetic and analytic study of neurosyphilis in its manifold aspects; and, as is proper, no less than one-third of the volume is devoted to questions of treatment. We note his hesitation as regards the vexed problem of neurotropic and dermotropic types of virus: his conclusion is against the dualistic theory. More significance attaches to the

mode of entry of the virus, though he feels it premature to assert categorically that the blood route leads to general paralysis and tabes, the subarachnoid to cerebrospinal syphilis. This view, however, will not commend itself to all, nor his contention that the encephalitis of the former affection is independent of accompanying syphilitic meningitis.

Although numerous authors are cited, the monograph lacks a list of bibliographical references, as well as an index.

Paralysie Générale et Malariathérapie. By R. LEROY, Medécin-chef à l'Asile Sainte-Anne, and G. MEDAKOVITCH, Chef de laboratoire à l'Asile Sainte-Anne. With an introduction by Prof. Dr. WAGNER-JAUREGG, Vienna. Paris: G. Doin et Cie. 1930. Pp. 500. Price 60 fr.

AN impressive work of no less than 500 pages, complete with bibliography (55 pages) and index, embraces all that is known, or worth knowing, of the malarial method of treatment for general paralysis. A succinct historical introduction reveals, what few perhaps have adequately realised, how definitely the idea of curing one ill by another was appreciated by certain of the forefathers of medicine. Of the technical details we need not here furnish any précis, but it is of significance to note the authors' decision that in cases where the malaria method fails, or has to be abandoned, supplementary treatment of an intensive kind with arsenicals, mercury and iodide in large doses proves highly efficacious. So far as we have observed, the one aspect of this wide-ranging subject which receives little attention is the question of the influence of natural malaria on neurosyphilitic development; otherwise, the monograph is replete with information and may be consulted with advantage by all who deal with the general paralytic in hospital or asylum.

Modern Psychotherapy. By EMANUEL MILLER, M.R.C.S., L.R.C.P., D.P.M., Honorary Psychopathologist, West End Hospital for Nervous Disease, etc. London: Jonathan Cape. 1930. Pp. 131. Price 5s. net.

THIS slender volume in the 'Modern Treatment Series' edited by Dr. F. G. Crookshank contains the meat of present-day psychotherapeutics with very little of the less digestible elements. Concerned largely with hypnosis, suggestion, re-education, as well as with the therapeutic doctrines of Freud, Jung, and Adler, it apprises the value of these respective methods, and, fortunately, indicates their limitations no less than their applications. The approach is purely psychological, and rightly so, although a short chapter on 'Psychotherapy in Organic Disease' is included; this, however, deals almost exclusively with the psychical depression of some chronic cases. The writer's pleasing literary style enhances the readable nature of his contribution.

Die zentrale Abstimmung der Sehsphäre. By Dr. HANS HOFF, Vienna. With 7 illustrations. Berlin: S. Karger. 1930. Pp. 96. Price R.M. 7.80.

THE purpose of this investigation is to determine to what extent and in what way or ways the function of a given region of the cerebral cortex is capable of being influenced by the function of areas in its vicinity or of other cortical mechanisms related to it physiologically. Choice is made of the visual cortex, and its connexions with the functions of sleep, of the vestibular apparatus, the sensory cortex, and so forth, are studied seriatim. Among other inferences from this study Dr. Hoff holds that with any sensory impression a whole series of centres are thrown into functional activity, and this renders it necessary to revise limited conceptions of cerebral localization. Contraction of a muscular group from electrical excitation of the motor area has little to do, in a sense, with a complete co-ordinated movement of a limb. It is here suggested that, in addition to their proper function, cortical 'centres' also act as 'points' at which impulses are braked, sent on, or diverted.

Techniques Histologiques de Neuropathologie. By IVAN BERTRAND, Chef de Laboratoire de la Clinique Neurologique de la Salpêtrière. With an introduction by Professor G. GUILLAIN. Paris: Masson et Cie. 1930. Pp. 375. Price 50 fr.

No laboratory of pathology or neuropathology should fail to acquire this histological compendium, written by a master of the art and embodying clear and practical descriptions of technical procedures of all the kinds in vogue to-day. It possesses a proper and adequate index and many references to original papers and other sources of information.

Die diagnostische Bedeutung des Rossolimoschen Reflexes. By Dr. S. GOLDFLAM, Warsaw. Berlin: S. Karger. 1930. Pp. 274.

IN 1902 Rossolimo described a reflex procedure of the following nature: with the patient lying on his back, limbs in extension, the plantar surface of the toes (toe pulp) is lightly but firmly stuck by the observer's fingers (held half-flexed) from below up: after a momentary diversion in a dorsal direction the toes contract with a definite flexion movement comparable to that of a tendon reflex, sometimes with a component of abduction also. This reflex was noted originally in cases of pyramidal involvement. To its study Dr. Goldflam devotes a monograph of more than 250 pages.

He finds it is highly characteristic of disseminated sclerosis, and may be demonstrated before a Babinski reflex develops: and he uses it by way of differential diagnosis between spinal compression and the former. He also states that absence of a Rossolimo reflex, with a positive Babinski response, suggests (in the case of hemiplegia) a capsular lesion: but the reverse is indicative of a cortical lesion. For numerous other clinical details the monograph should be consulted.

Klinisch en experimenteel Onderzoek over Monocytose bij Aandoening der Groote Hersenen. By Dr. R. GEERLING, Amsterdam. Amsterdam: Scheltema and Holkema. 1930. Pp. 131. Price not stated.

IN nine cases of deeply placed frontal tumours Dr. Geerling found a monocytosis in the blood (large mononuclears) ranging round 10 per cent. or higher, whereas in a series of tumours localised elsewhere no such increase in percentage was seen. A number of experimental lesions of destructive nature and varying size were made in rabbits and their blood subsequently examined. The conclusion is, that in bilateral lesions of the frontal lobes, provided they are deep enough to invade the basal ganglia, a relative monocytosis follows.

Clinical Examination of the Nervous System. By G. H. MONRAD KROHN, M.D., F.R.C.P. Fifth edition. London: H. K. Lewis & Co. 1930. Pp. 202. Price 7s. 6d. net.

THE fifth edition of this now well-known handbook contains but little new matter. Short accounts have been added on the uses and technique of ventriculography and encephalography and on the more recent methods for the employment of hypertonic solutions for the temporary relief of increased intracranial tension. As a concise guide to the examination of the nervous system it can be confidently recommended.

Psychiatrie. By ANDRÉ BARBÉ, Medécin aliéniste des Hôpitaux de Paris. With a preface by Dr. J. SEGLAS. Paris: Masson et Cie. 1930. Pp. 195. Price 16 fr.

THIS little vade mecum is for the student entering on psychiatry and is composed of three divisions, dealing respectively with the patient's symptoms, his examination, and the pathogenesis of mental affections. Within its limits it serves a useful purpose.

The Physiological Principles of Hydrology. By R. G. GORDON, M.D., D.Sc., F.R.C.P., and F. G. THOMSON, M.A., M.D., F.R.C.P. London: Jonathan Cape. 1930. Price 5s. net.

THIS little member of the 'Modern Treatment Series' will be welcome to many members of the profession whose knowledge of spa treatment is only second-hand.

It provides an admirable résumé not only of the principles on which treatment by hydrological methods should be based but also of the different methods available and their appropriate uses. The authors have been careful to make no undue claims for the benefits to be obtained from spa treatment but they have succeeded in indicating clearly the proper rôle which such treatment can play in the relief of the many disorders for which the internal administration or the external application of mineral waters may properly be recommended.

Physiotherapy: its Principles and Practice. By F. HOWARD HUMPHRIS, M.D., F.R.C.P.Ed., etc., and R. E. STUART-WEBB, M.B. With special contributions by other writers. London: Jonathan Cape. 1930. Pp. 384. Price 15s. net.

THERE is much of direct interest to the neurologist in this compilation, but not enough. The nervous disease-conditions specifically discussed from a physiotherapeutic view embrace hemiplegia, tabes, infantile paralysis, disseminated sclerosis, neuritis, neurasthenia, headache, migraine, tic douloureux, and insomnia. Though a special section is assigned to radium we find no mention of it, or of X-ray therapy, in the treatment of tumours of the neuraxis, while there are other nervous conditions not here included in which physiotherapy of one or other kind justifies its adoption, or is worthy of trial (syringomyelia, hysteria, torticollis, forms of occupation neurosis, etc.). The book is so good as a whole, and so well-written, that we may ignore the authors' little trips into the field of neuropathology with which it is evident they are not entirely familiar.